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Ambient air pollution in Massachusetts: inequality trends, residential infiltration, and childhood weight growth trajectories

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AMBIENT AIR POLLUTION IN MASSACHUSETTS:

INEQUALITY TRENDS, RESIDENTIAL INFILTRATION,

AND CHILDHOOD WEIGHT GROWTH TRAJECTORIES

by

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DEDICATION

Dedicated to my parents, Laurie and Robert, my brother, Gabriel, and to my fiancé, Jonathan.

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ABSTRACT

Exposure to pollutants of ambient origin contributes significantly to the global disease burden (Cohen et al., 2017). Mounting evidence has demonstrated disproportionately high ambient $PM_{2.5}$ and NO_2 concentrations in the U.S. among nonwhite and low-income populations, potentially contributing to environmental health disparities (Bell and Ebisu, 2012; Clark et al., 2014; Morello-Frosch and Lopez, 2006). There is limited understanding of temporal trends and underlying causes of exposure inequalities (EIs), and whether residential building characteristics modify observed EIs. Further, while ambient pollutants have been linked to cardiometabolic disease in adulthood, few studies have documented the link between early-life ambient air pollution exposure and weight growth trajectories in early childhood- an informative step on the causal pathway between early life exposures and chronic outcomes.

Using 1 km^2 $PM_{2.5}$ and NO_2 predictions in Massachusetts and Census data, we quantify longitudinal EI between sociodemographic groups over a decade. We estimate AER for all Massachusetts residential parcels using publicly available data and assess whether accounting for AER exacerbates or ameliorates $PM_{2.5}$ inequalities. We examine

associations of weight growth trajectories in early childhood with residential prenatal and postnatal $PM_{2.5}$ and distance to road (traffic) exposure in the Boston-based Children's HealthWatch cohort.

$PM_{2.5}$ and NO_2 inequalities increased across the study period in urban areas, and EIs were more pronounced for NO_2 than $PM_{2.5}$ and among racial/ethnic groups compared to other population subgroups. Analyzing EI longitudinally revealed that spatio-temporal shifts in air pollution, and not demographic distributions, contributed to exposure disparities. We found substantial variability in estimated AER across the state, and that $PM_{2.5}$ EIs were magnified when AER was considered. Prenatal $PM_{2.5}$ >9.5 $\mu g/m^3$ predicted higher weight growth rates among females, but with an opposite direction of effect in males. This association was modified by birth weight and AER, with a stronger magnitude of effect in low-birthweight and higher-AER females.

These findings underscore the importance of considering vulnerable communities and residential characteristics in ambient air pollution reduction strategies. This dissertation provides an opportunity to understand susceptible phenotypes and periods of potential intervention to reduce ambient air pollution impacts on cardiometabolic outcomes.

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CHAPTER 1. INTRODUCTION

Exposure to outdoor ambient air pollution is ubiquitous and a growing public health concern. According to the Global Burden of Disease, ambient air pollution is the 5th leading cause of mortality in the world (Cohen et al., 2017). Elevated concentrations of particulate matter less than or equal to 2.5 μm in aerodynamic diameter ($PM_{2.5}$) and nitrogen dioxide (NO_2) contribute to global morbidities over the life course. $PM_{2.5}$ and NO_2 exposure during the prenatal periods has been linked to adverse birth outcomes (Brauer et al., 2008), such as low birth weight and short gestational age, increased childhood asthma morbidity and severity and respiratory infections (Brauer et al., 2002), neurodevelopmental disorders (Volk et al., 2013a), and metabolic dysregulation during childhood (Calderón-garcidueñas et al., 2015). Chronic postnatal exposure has been documented as a risk factor for cardiovascular diseases (Hart et al., 2015; Lee et al., 2014; Zanobetti et al., 2000), obesity (Jerrett et al., 2014) and associated disorders such as increased adiposity and type 2 diabetes, and all-cause mortality (Faustini et al., 2014; Franklin et al., 2008; Shi et al., 2016). Many of these health effects have been observed at concentrations below the National Ambient Air Quality Standards set by the Clean Air Act of 12 $\mu g/m^3$ for $PM_{2.5}$ and 53 ppb for NO_2 (Franklin et al., 2007; Shi et al., 2016).

NO_2 is a primary byproduct of traffic pollution. Nitric oxide (NO) is emitted primarily from automobile exhaust and other fuel combustion processes. Once released into the atmosphere it combines with oxygen to produce NO_2. NO_2 concentrations exponentially decline with distance from their source, and are therefore used as a marker of local source pollutants, particularly road traffic (Zhu et al., 2002). Several different regional sources contribute to ambient $PM_{2.5}$ concentrations, including wood burning, industrial processes, fuel combustion and mobile sources. $PM_{2.5}$ components that are directly emitted from the aforementioned sources include heavy metals, sulfate, sodium chloride, and elemental carbon. $PM_{2.5}$ constituents that are formed via secondary processes in the atmosphere include ammonium nitrate, ammonium sulfate and secondary aerosols. $PM_{2.5}$ is distributed regionally and from a wider variety of sources than NO_2 (Karner et al., 2010; Zhou and Levy, 2007).

Inequitable Distribution of Ambient Air Pollutants

Since the publication of *Toxic Wastes and Race* in 1987, documenting disproportionate siting of uncontrolled toxic waste sites in Black and Hispanic communities, evidence has accumulated demonstrating inequitable distribution of sources and concentrations of a variety of environmental contaminants by age, race and ethnicity, education and income. Ambient $PM_{2.5}$ and NO_2 are two such pollutants for which disproportionately high concentrations among non-white and low-income groups has been extensively documented (Bell and Ebisu, 2012; Clark et al., 2014; Morello-Frosch and Lopez, 2006; Su et al., 2009). Social stressors associated with neighborhood and residential characteristics, such as poverty, poor housing quality, racial discrimination, and limited access to health resources affect these same communities (Morello-Frosch et al., 2011). The cumulative burden of environmental exposures and social stressors have important implications for health outcomes disproportionately prevalent in these communities, such as hypertension, diabetes, asthma and preterm birth (Lopez, 2002; Morello-Frosch and Lopez, 2006; Morello-Frosch and Shenassa, 2006).

The ability for existing environmental inequality studies to accurately assess spatial distribution and underlying drivers of inequalities is limited by existing methodologies. Spatial coincidence and proximity-based methods are commonly used to assess inequality (Chakraborty et al., 2011; Legot et al., 2012; Pastor et al., 2004). Spatial coincidence methods compare demographic characteristics between geographic units (counties, zip codes, Census tracts, etc.) containing varying counts of pollution sources or concentrations within a given unit. Proximity-based approaches compare demographic characteristics of geographic units at different spatial buffers from a given pollution source. Although these methods are useful to examine general correlations between source locations and demographic distributions, they do not ultimately reflect population exposures, as they ignore chemical fate and transport and local meteorological conditions. These limitations make identifying environmental inequalities occurring at smaller spatial scales challenging. A growing number of exposure inequality studies have improved upon these methods by using modeled or measured ambient concentrations, but these are at relatively coarse resolutions and are performed cross-sectionally (Clark et al., 2014; Morello-Frosch and Jesdale, 2006; Pope et al., 2016).

Regardless of the method, most environmental inequality research to date has examined inequalities at one point-in-time (Legot et al., 2012; Mohai et al., 2011; Pastor et al., 2004). While these studies have been essential to establish the existence of environmental inequality in the United States, they do not document whether patterns of inequality have remained steady, increased or decreased over time. Longitudinal environmental inequality studies not only inform our understanding of general patterns of exposure, but also provide insights into relationships between health and exposure disparities, and whether national and regional policies aimed at decreasing overall $PM_{2.5}$ and NO_2 concentrations have benefited populations subgroups disproportionately.

Longitudinal studies also allow for an understanding of the mechanisms that drive exposure inequalities. There is substantial disagreement about whether exposure inequalities are a consequence of disproportionate siting near vulnerable communities, demographic change around existing pollutant sources, or both (Mohai and Saha, 2015a). Much of this disagreement may be attributed to the lack of longitudinal studies that have examined exposure inequality over time. Mohai and Saha (2015) conducted a review of the cross-sectional and the handful of longitudinal studies available at the time of publication and outlined three principal themes that drive inequitable siting and population movement: economic explanations (siting near low-income communities is cheaper for business), sociopolitical explanations (siting near disempowered communities), and segregation due to racial discrimination. In their conclusions, the authors state: "To bring greater clarity to how environmental disparities come about, we thus believe longitudinal studies that employ ecological (census) data will also continue to be needed along with studies that use individual/household level (survey) data." Since the publication of the above review, only two U.S.-based studies have explored temporal trends in ambient air pollution exposure inequality (Clark et al., 2017; Kravitz-Wirtz et al., 2016). Both found that despite overall decreasing trends in air pollution, exposures remained higher for nonwhite compared to white populations (Clark et al., 2017; Kravitz-Wirtz et al., 2016). More longitudinal inequality studies of ambient pollutants are needed to illuminate the origins of these inequalities.

Effects of Residential Characteristics on Personal Ambient Air Pollution Exposure

The home environment is closely linked to both residential segregation and poverty (Rauh et al., 2008). Investigators in both the sociological and environmental health literature argue that residential segregation, and in turn, individual residential environments, are main drivers in the persistence of environmental health disparities (Mohai and Saha, 2015; Morello-Frosch and Lopez, 2006). Structural mechanisms of housing discrimination impact both household and community-level determinants of environmental pollution and ultimately disease burden. For instance, low-income residents often live in smaller and older units, resulting in different household-level ventilation patterns that may contribute to the persistence of disparities in health (Adamkiewicz et al., 2011).

Institutional factors influence both the housing stock and neighborhood contribution to ambient pollution. In addition to lack of policy that incorporates environmental justice in industrial siting and residential proximity to traffic, inadequate city code enforcement and residential instability may lead to deterioration in the neighborhood housing stock and value. The extent to which the inequitable distribution of ambient pollution overlaid on social-structural conditions that further exacerbate exposure inequities needs to be further explored.

Air pollution epidemiology and exposure inequality studies typically use ambient concentrations as surrogates for personal exposure, which fail to capture inter-individual exposure variability (Williams et al., 2003). Individuals in the United States spend approximately 90% of their time indoors and 70% of their time in the homes (Klepeis et al., 2001). Therefore, indoor exposure to pollutants of ambient origin are an important component of an individual's overall exposure, and the individual and community level housing stock plays an important role in modifying the extent to which these pollutants enter the indoor environment. Indoor concentrations of outdoor ambient NO_2 and $PM_{2.5}$ are of particular concern in low-income communities that are often faced with poor housing stock and higher asthma prevalence and severity (Fabian et al., 2014, 2012b). Personal exposure monitors may be used to capture exposure variability from differential micro-environmental conditions, including in the residential environment (Jedrychowski et al., 2009; Ozkaynak et al., 1996; Perera et al., 2013), but they are rarely implemented in epidemiologic and inequality studies because they are both intrusive and costly. Alternative and efficient methods that can be implemented in large-scale studies are needed to capture physical characteristics of the home that bring about exposure variability of ambient air pollutants.

Outdoor ambient particle infiltration into the home environment is determined by factors that drive air exchange rate (AER) which in turn is governed by three main processes: mechanical ventilation (HVAC systems and kitchen vents), natural ventilation (window opening), and infiltration (home leakiness) (Breen et al., 2013). In this study, we focus exclusively on mechanisms of infiltration that impact ambient air pollution exposure. Tracer gas measurements are typically used to estimate AER but are time and labor-intensive. Alternatively, AER can be estimated using physical or empirical-based methods, but these too require detailed data collection of physical building characteristics, meteorological conditions, surrounding terrain and building density, and age of the home (Breen et al., 2013). These limitation resulted in a limited number of studies incorporating characteristics of the residential environment into air pollution exposure estimates (Baxter et al., 2007; Chen et al., 2012; Levy et al., 2005; Ozkaynak et al., 2013; Sarnat et al., 2013; Zota et al., 2005).

Because many environmental inequality studies use easily accessible data over large populations and spatial scales, a straightforward method that estimates AER using publicly-available data is needed to incorporate AER into future exposure inequality studies. Only one study to date has used publicly-available data to measure how geographic and temporal variations in residential AER modifies the association between ambient CO, NOx, O_3 and $PM_{2.5}$ and a health outcome over a large region (Sarnat et al., 2013). In this study, the authors found that, on the zip code level, daily variability in AER within a single-city time series model may explain heterogeneity in longitudinal asthma emergency department visits. Pollutant-related asthma emergency department visits were significantly higher on days with higher modeled AER, demonstrating that AER does modify health outcomes (Sarnat et al., 2013).

Ambient Air Pollution and Childhood Growth Trajectories

The prevalence of childhood obesity and adiposity in children is a prominent public health concern in the U.S. (Skinner and Skelton, 2014). Childhood obesity is a strong determinant of a range of morbidities associated with metabolic dysregulation in adulthood, including type 2 diabetes, poor cardiovascular outcomes and hypertension (Barker et al., 2005; Matthews et al., 2017; The GBD 2015 Obesity Collaborators, 2017).

While dietary and genetic factors are important determinants of childhood obesity and metabolic disorders throughout the life course, a growing body of literature is demonstrating links between ambient air pollution exposure and excess weight gain in childhood (Fleisch et al., 2016, 2015, Jerrett et al., 2014, 2010; Mao et al., 2016; McConnell et al., 2015).

There is an extensive body of literature on prenatal air pollution and birthweight, a known risk factor for many of the cardiometabolic disorders listed above (Zheng et al., 2016). However, the continuing effects of prenatal $PM_{2.5}$ exposure on the postnatal growth trajectories are not well established. To address this gap, a limited number of studies have assessed prenatal and chronic exposure to $PM_{2.5}$ and traffic exposure with attained measures of metabolic outcomes (birth weight, raw weight, BMI, adiposity) at various life stages (Chiu et al., 2017; Fleisch et al., 2015; McConnell et al., 2015). In a Boston-area pregnancy cohort, Fleisch et al. (2015) found an increased odds of weight-for-length >95th percentile at 6 months of age in 4th quartile third-trimester $PM_{2.5}$ (β =-0.08 units, 95% CI: -0.2, 0.04) and distance to roadway < 50 m compared to the referent group, though estimates were imprecise. In a follow-up study, IQR increase in postnatal one-year average $PM_{2.5}$ concentrations prior to each measurement occasion was associated with lower BMI-z score, total and truncal fat mass in mid-childhood (average 8 years of age) (Fleisch et al., 2016). Mao et al. (2016) found an increased risk of overweight (BMI z-score >= 85th percentile) and obesity (BMI z-score >= 95th percentile) in ages 2-9 years in the highest vs. lowest quartile of average prenatal $PM_{2.5}$ exposure (OR=1.3 (95% CI: 1.1, 1.6)) and postnatal $PM_{2.5}$ in the first two year of life (OR=1.2

(95% CI:1.1, 1.5)) (Mao et al., 2016).

There are some hypothesized biological mechanisms explaining these associations. Studies in rodent models have suggested that prenatal $PM_{2.5}$ exposure may cause intrauterine inflammation and oxidative stress (Bolton et al., 2012; Sun et al., 2009). Oxidative stress in the fetal environment may interfere with transcription of genes known to maintain cardiovascular and metabolic homeostasis (Keane et al., 2015; Thompson and Al-Hasan, 2012). Other studies have found that prenatal $PM_{2.5}$ exposure may be associated with epigenetic mechanisms that alter specific genes associated with regulating growth and satiety in adulthood, such as leptin methylation (Bolton et al., 2012; Calderón-Garcidueñas et al., 2014; Chen et al., 2017; Horvath, 2005). Other human and rodent studies have demonstrated a biological relationship between postnatal air pollution exposures and metabolic syndrome in children and in adulthood, including increased risk of insulin resistance and blood markers of inflammation (Brocato et al., 2014; Brook et al., 2010; Eze et al., 2015; Xu et al., 2010).

The vast majority of studies examining pre- and postnatal $PM_{2.5}$ and traffic exposure have focused almost exclusively on prenatal exposures and weight outcomes measured cross-sectionally (Zheng et al., 2016). A remaining literature gap are studies identifying the periods at which different growth trajectories diverge with varying levels of postnatal ambient air pollution exposure. Risk of metabolic disorders in adulthood has been linked with specific longitudinal growth phenotypes in early childhood (Eriksson, 2011; Vaag et al., 2012). However, the research investigating early life social and environmental factors that shape long-term weight trajectories is still in nascent stages

(Braun et al., 2016; Howe et al., 2015, 2012; Johnson et al., 2014; Tilling et al., 2014). Following growth trajectories longitudinally can help identify periods of intervention to mitigate the impacts of air pollution exposure, and provide an opportunity to more comprehensively investigate the link between exposures and growing rates of obesity.

To our knowledge, only one study to date has investigated associations between continuous longitudinal postnatal growth trajectories and air pollution (Jerrett et al., 2014). In this study, postnatal traffic density within 150 meters of the home was not significantly associated with BMI growth rates between the ages of 5 and 11 years of age in adjusted models. The authors did find a significant increase in BMI growth rates with NOx in adjusted models (0.087 BMI (kg/m^2) per year per unit NOx) (Jerrett et al., 2014). More research that examines air pollution effects of early childhood weight trajectories longitudinally is needed to elucidate the mechanisms on the causal pathway between early-life air pollution exposure and metabolic disorders later in life.

Dissertation Objectives

To address the data gaps outlined above, this dissertation addresses three specific objectives, explained in more detail below:

1) Characterize longitudinal $PM_{2.5}$ and NO_2 exposure inequality across Massachusetts (Chapter 2);
2) Estimate AER across all Massachusetts residential parcels to examine the spatial distribution of home leakiness and to use these AER estimates to analyze exposure inequalities across sociodemographic subgroups (Chapter 3);

3) Examine associations between prenatal and postnatal ambient $PM_{2.5}$ and traffic exposure and longitudinal weight growth trajectories in early childhood (Chapter 4).

Objective 1 (Chapter 2):

Though air pollution exposure inequality literature has demonstrated compelling evidence of disproportionate air pollution exposures among low-income and ethnic minority communities across the U.S., it is unclear whether these inequalities have intensified or declined while U.S. air pollution policy has strengthened over time and issues of exposure inequality have come to light.

In this chapter, we characterize longitudinal inequality trends in modelled $PM_{2.5}$ and NO_2 concentrations between demographic subgroups across Massachusetts. A sub-aim of this paper is to understand whether observed longitudinal patterns in exposure inequalities are driven by population mobility or spatial distribution of ambient pollutants. To address this question, we hold demographic data constant with changing annual $PM_{2.5}$ and NO_2 concentrations to determine whether population mobility or differential spatial variability in air pollution concentrations drive observed inequalities.

Objective 2 (Chapter 3):

The goals of this chapter are twofold: 1) to develop a flexible measure of home-leakiness that could be applied in ambient air pollution exposure studies; 2) to assess whether variability in residential characteristics that affect infiltration of ambient pollutants indoors exacerbates or ameliorates exposure inequalities. Using the validated physical and empirical-based Lawrence Berkeley Laboratory (LBL) AER model and publicly-available housing, land-use and meteorological data, we estimate AER for all residential parcels in Massachusetts. We then calculate indoor $PM_{2.5}$ concentrations of ambient origin to assess whether $PM_{2.5}$ concentration variability increases when residential characteristics are considered.

To evaluate whether $PM_{2.5}$ exposure inequality is modified by AER, we identify the parcels in the 10^{th} and 90^{th} percentiles of AER, of $PM_{2.5}$, or of both AER and $PM_{2.5}$. We then overlay these parcels on Census block groups and examine the demographic distributions of the block groups in which these identified parcels are located. We hypothesize that exposure inequalities are magnified in the analysis of combined $PM_{2.5}$ and AER distributions, compared to the distributions separately. In this work, we demonstrate an efficient and flexible method to apply AER to future studies involving estimates of personal ambient air pollution exposure.

Objective 3 (Chapter 4):

In this chapter, we examine associations between prenatal and postnatal $PM_{2.5}$ concentrations and residential distance to major roadways with postnatal early-childhood weight-for-age growth trajectories of participants in the Children's HealthWatch (CHW) cohort. Boston CHW is an open sentinel surveillance study of multiethnic caregivers who access child health care at Boston Medical Center (Frank et al., 2013). This work is restricted to Boston CHW participants enrolled in CHW between 2009 and 2015.

Using a robust set of individual and neighborhood-level covariates derived from CHW surveys and EMRs, we create multivariable mixed models to assess the link between longitudinal weight growth trajectories and three exposures of interest: average prenatal ambient $PM_{2.5}$, residential distance to major road at each weight observation, and postnatal rolling-average ambient $PM_{2.5}$. We stratify all models by sex and assess effect modification by birth weight and the AER values estimated in *Chapter 2.* This research aims to broaden understanding of the steps on the causal pathway between early-life air pollution exposure and metabolic disorders later in life.

Chapter 5 summarizes the findings of *Chapters 2–4*, as well as a discussion of shared limitations across *Chapters 2-4,* overall public health implications and directions for future research.

CHAPTER 2. TEMPORAL TRENDS IN AIR POLLUTION EXPOSURE INEQUALITY IN MASSACHUSETTS

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Abstract

Mounting evidence over the past several decades has demonstrated inequitable distribution of pollutants of ambient origin between sociodemographic groups in the United States. Most environmental inequality studies to date are cross-sectional and used proximity-based methods rather than modeled air pollution concentrations, limiting the ability to examine trends over time or the factors that drive exposure inequalities. In this paper, we use 1 km^2 modeled $PM_{2.5}$ and NO_2 concentrations in Massachusetts over an 8-year period and Census demographic data to quantify inequality between sociodemographic groups and to develop a more nuanced understanding of the drivers and trends in longitudinal air pollution inequality.

Annual-average population-weighted $PM_{2.5}$ and NO_2 concentrations were highest for urban non-Hispanic black populations (11.8 $\mu g/m^3$ in 2003 and 8.4 $\mu g/m^3$ in 2010, vs. 11.3 $\mu g/m^3$ and 8.1 $\mu g/m^3$ for urban non-Hispanic whites) and urban Hispanic populations (15.9 ppb in 2005 and 13.0 ppb in 2010, vs. 13.0 ppb and 10.2 ppb for urban non-Hispanic whites), respectively. While population groups experienced similar absolute decreases in exposure over time, disparities in population-weighted concentrations increased over time when quantified by the Atkinson Index, a relative inequality measure. Exposure inequalities were approximately one order of magnitude greater for NO_2 compared to $PM_{2.5}$, were more pronounced in urban compared to rural geographies, and between racial/ethnic groups compared to income and educational attainment groups. Our results also revealed similar longitudinal $PM_{2.5}$ and NO_2 inequality trends using Census 2000 and Census 2010 data, indicating that spatio-temporal shifts in air pollution may best explain observed trends in inequality. These findings enhance our understanding of factors that contribute to persistent inequalities and underscore the importance of targeted exposure reduction strategies aimed at vulnerable populations and neighborhoods.

Keywords: air pollution; environmental inequality; environmental justice; longitudinal analysis; inequality index

Background

Ambient exposure to nitrogen dioxide (NO_2) and fine particulate matter ($PM_{2.5}$) have been associated with a range of adverse health effects. These include increased risk of asthma and respiratory infections (Brauer et al., 2002; O'Connor et al., 2008; Xing et al., 2016), adverse birth outcomes such as early gestational age and low birth weight (Brauer et al., 2008; Stieb et al., 2012; Zheng et al., 2016), increased risk of autism spectrum disorders (Raz et al., 2015; Volk et al., 2013b), and all-cause mortality (Franklin et al., 2008; Shi et al., 2016). Mounting evidence over the past several decades has demonstrated inequitable distribution of exposure to $PM_{2.5}$ and NO_2 in the United States among children and older adults, non-Hispanic black and Hispanic populations, low educational attainment and low income populations, potentially contributing to environmental health disparities (Bell and Ebisu, 2012; Brugge et al., 2015; Clark et al., 2014; Morello-Frosch and Lopez, 2006; Su et al., 2009).

However, there are three key limitations in the exposure inequality literature to date. First, much of the environmental inequality (EI) research is cross-sectional, examining environmental inequalities at one point in time. This limits the ability to examine longitudinal trends or the causal mechanisms that drive inequality (Legot et al., 2012; Mohai et al., 2011; Pastor et al., 2004). In particular, there is limited insight about whether disparities are driven by population shifts subsequent to siting of hazardous facilities or roadways, disparate siting practices in poor communities and communities of color, or policies focused on decreasing ambient pollution that simply do not examine distributional consequences. Investigators in both the sociological and environmental health literature argue that residential segregation is a main driver of environmental health disparities (Mohai and Saha, 2015; Morello-Frosch and Lopez, 2006). Demographic shifts over time could have an influence on land use practices, declining social capital and local economies and ultimately, community-level environmental exposures (Mohai and Saha, 2015b; Pastor et al., 2004, 2001). Further, demographic change over time could modify inequalities even in the absence of changes in air quality. Therefore, it is imperative to incorporate demographic time trends in air pollution exposure inequality studies.

Second, a limited number of studies have used quantitative metrics to assess EI over space and time. Quantifiable measures of exposure inequality allow regulators to formally assess patterns of EI and to maximize efficiency in exposure reduction policies that seek to reduce environmental exposures, while simultaneously incorporating social equity into distributional assumptions (Boyce et al., 2016; Harper et al., 2013; Levy et al., 2007, 2006). A handful of environmental studies to date have incorporated formal inequality indices to assess geographic and social distribution of environmental hazards (Boyce et al., 2016; Clark et al., 2014; Levy et al., 2007, 2006; Post et al., 2011; Su et al., 2009). These previous studies have adopted welfare-based or health-based measures of inequality to assess sociodemographic distributions of exposure to a single hazard (Boyce et al., 2016; Clark et al., 2014; Fann et al., 2011; Levy et al., 2006; Post et al., 2011) or cumulative environmental hazards (Su et al., 2009). This paper employs the Atkinson Index (AI) (Atkinson, 1970), a relative measure of inequality, discussed in further detail below. Although some previous studies have used the AI to quantify exposure inequality

(Clark et al., 2014; Levy et al., 2009, 2006; Post et al., 2011), these studies focused to a greater extent on understanding the inequality implications of air pollution control strategies, and not on longitudinal patterns of inequality.

Most EI studies examine inequitable distributions of hazardous facilities among population subgroups (Mohai and Saha, 2015a, 2015b). A limited, but growing number of EI studies have examined inequalities with respect to both hazardous facilities and traffic-related air pollution using modeled or measured ambient concentrations. However, many are at coarse geographic resolutions, ignore chemical fate and transport and local meteorological conditions, and do not address longitudinal trends in EI (Clark et al., 2014; Hajat et al., 2015; Kravitz-Wirtz et al., 2016; Mohai and Saha, 2015a; Morello-Frosch and Jesdale, 2006; Pope et al., 2016). Pollutants such as NO_2 and $PM_{2.5}$ have significant public health burdens but are not typically dominated by local emissions from hazardous facilities, reinforcing the importance of an exposure-based analytical approach to identify EI occurring at smaller spatial scales.

In this paper, we quantify inequality in modeled ambient $PM_{2.5}$ and NO_2 concentrations between racial, ethnic, income and education groups across Massachusetts between 2003 and 2010 using methods to address the three major limitations in this area of research. The work applies a formal inequality index to examine patterns of exposure among rural and urban populations as a means to identify populations most vulnerable to air pollution exposure within the state. The availability of demographic data from the decennial 2000 and 2010 Census at the block group level and modeled ambient air pollution at a 1 km^2 resolution over an eight-year period provides us the unique opportunity to examine inequalities over time and develop a more nuanced understanding of whether $PM_{2.5}$ and NO_2 exposure inequalities are driven by demographic shifts or longitudinal pollution source distribution.

Methods

Data Sources

Ambient air pollution for Massachusetts, 2003-2010. Daily surface $PM_{2.5}$ at a 1 km^2 resolution was modeled from 2003-2010 using a 3-stage statistical modeling approach (Kloog et al., 2014). This modeling approach used a combination of aerosol optical depth (AOD) satellite data retrieved using the multi-angle implementation of atmospheric correction (MAIAC) algorithm, land use and meteorological predictors of variation in surface-$PM_{2.5}$, and monitored $PM_{2.5}$ concentrations (Kloog et al., 2014). This produced an overall "out-of-sample" R^2 for daily values of 0.88, and cross validation results produced a slope of observed versus predicted of 0.99. Details of the $PM_{2.5}$ prediction models can be found in in Kloog et al. (2014).

We used daily ground NO_2 concentrations that were estimated for the New England region from 2005-2010 at a 1 km^2 resolution from a combination of ground-level NO_2 data at monitoring sites, satellite Ozone Monitoring Instrument NO_2 vertical column density data, and land use regression (Lee and Koutrakis, 2014). Predictors in mixed effects models included population density, distance to major highways, percent developed area, NO_2 source emissions, elevation, and temperature data. This model produced an R^2 of 0.79 and cross validation results produced a slope of observed versus predicted of 0.98, demonstrating high predictive reliability. NO_2 model details can be found in Lee and Koutrakis (2014).

Demographic Data. We gathered geographic distributions of race/ethnicity, income, and educational attainment from the US Census and American Community Survey (ACS) at the block group unit of analysis. Measures of educational attainment and income were not collected in the decennial 2010 Census. Therefore, we obtained race/ethnicity data from Census 2010, and measures of income and educational attainment from ACS 2006-2010 5-year estimates. We categorized block groups as rural and urbanized centers according to Census classifications, which rely on population density (Ratcliffe et al., 2016). We utilize Census data at two distinct time periods, 2000 and 2010, rather than at 1-year intervals over the decade under study because the non-decennial 1-year summaries from the ACS are less-reliable, constitute a smaller sample size, and were only collected starting in 2005.

We categorized population characteristics into the following groups:

- Race/ethnicity: individuals in each block group that self-identify as non-Hispanic white, non-Hispanic Black, non-Hispanic Asian, Hispanic or other
- Income: 1999 and 2010 inflation-adjusted median household income as <$20,000/year, $20-35,000/year, $35-50,000/year, $50-75,000/year, and >$75,000/year
- Educational Attainment: individuals in each block group ≥25 years of age with less than a high school degree, high school graduate, postsecondary degree, bachelors and graduate degree

We aggregated daily $PM_{2.5}$ and NO_2 concentrations to average annual concentrations. Annual $PM_{2.5}$ (years 2003-2010) and NO_2 (years 2005-2010) concentrations were assigned to each block group centroid using the closest 1 km^2 grid cell centroid for each year over the study period. This exposure assignment method was performed separately for Census 2000 and ACS/Census 2010 block groups using ArcGIS 10.3 (ESRI, Inc.).

Statistical Analysis

Summary Statistics. We calculated summary statistics for Massachusetts of the number and percentage of individuals and households within each racial/ethnic and education group and the percentage change between 2000 and 2010 stratified by urban (densely developed territories with 50,000 or more people (Census 2000, n=4277; Census and ACS 2010, n=4308)) and rural (any territory not defined as urban (Census 2000, n=654; Census and ACS 2010, n=596)) block groups (Table 2.1). We also present median household income in 2010 dollars for both time points. Due to the small number of block groups categorized by the Census Bureau as "urban clusters," territories containing between 2,500 and 50,000 residents (Census 2000, n=116; Census and ACS 2010, n=75), these block groups were excluded from stratified analyses.

Calculating Population-Weighted Concentrations across Subpopulations. We calculate block group population-weighted $PM_{2.5}$ concentrations for each year from 2003 to 2010, and population-weighted NO_2 concentrations for each year from 2005 to 2010. As corresponding annual population data are not available, we calculate separately using each of Census 2000, Census 2010 and ACS 2006-2010, and we evaluate the influence of using alternative population data. Population-weighted concentrations were calculated for each population demographic group stratified by urban and rural classifications. Block-group $PM_{2.5}$ and NO_2 ($PM_{2.5i}$, NO_{2i}) were multiplied by the number of people in each population subgroup (p_i). The subgroup block group values were summed for the state and divided by the total population in each subgroup:

$$\frac{\sum PM_{2.5_i} p_i}{\sum p_i} \quad \text{or} \quad \frac{\sum NO_{2_i} p_i}{\sum p_i} \qquad \text{[Equation 2.1]}$$

Quantifying Inequality Using the AI. We formally quantify air pollution exposure inequality between population subgroups using the Atkinson Index (AI) (Atkinson, 1970). The AI has been traditionally used as a welfare-based measure of income inequality (Kawachi and Kennedy, 1997), but has since been adopted in the environmental inequality literature (Clark et al., 2014; Levy et al., 2007, 2006). The AI can be decomposed into between-group, within-group and total inequality measures (Lasso de la Vega and Urrutia, 2003; Levy et al., 2006). This feature allows investigators to compare distributions of pollutants between population subgroups, and determine whether total inequality in a population can be explained by disproportionate pollution burden between population subgroups (Harper et al., 2013).

For the purposes of the current analysis, we present only "between-group" inequality, as the focus of this paper is examining trends in environmental inequality between population subgroups given the environmental justice implications. The AI ranges from zero to one, zero indicating no inequality and one indicating complete inequality.

The Between-Group AI can be expressed as:

$$1 - \left(\sum_{j=1}^{n} f_j \left[\frac{\bar{y}_j}{\bar{y}}\right]^{1-\varepsilon} \right)^{\frac{1}{1-\varepsilon}} \qquad \text{[Equation 2.2]}$$

where n represents the number of individuals in the population, f_j represents the fraction of the total population in each subgroup, $\bar{y}_j$ represents mean exposure of each subgroup, $\bar{y}$ represents the mean exposure over the full population within a given geographic boundary (state, rural or urban) and ε represents an explicit inequality aversion parameter, explained below (Atkinson, 1970). The AI is therefore a relative (as opposed to absolute) measure of inequality, so that proportional changes in exposure across the population would not influence the AI, but additive changes would have an effect. By comparing each subgroup's weighted exposure to the overall population average exposure within a defined geography, the between-group AI represents the magnitude, in relative terms, of exposure disparities between population subgroups. This is an overall measure of inequality between defined subgroups- it does not explicitly provide information about which of those subgroups are most inequitably exposed.

The inequality aversion parameter is a measure of societal concern about inequality. It determines where relative weights should be placed across the exposure distribution. The parameter ranges from zero to infinity, with increasing values reflecting greater weight on the bottom of the distribution. Unlike income, environmental exposures are worse at higher levels, so we perform all AI calculations with the inverse of the pollution concentrations to allow for interpretable calculations (Harper et al., 2013). All calculations presented here apply an aversion parameter of 0.75, consistent with the literature (Clark et al., 2014; Fann et al., 2011; Levy et al., 2009, 2007, 2006; Post et al., 2011). As a sensitivity analysis, we report the AI for multiple alternative inequality aversion parameters (0.25-2) in the Appendix (Figure S2.4).

Applying the AI (ε=0.75), we quantified between-group $PM_{2.5}$ and NO_2 exposure inequality by applying average annual $PM_{2.5}$ concentrations for each year between 2003 and 2010 and average annual NO_2 concentrations for each year between 2005 and 2010, keeping the demographic data constant.

Results

Population Characteristics

State-wide demographic characteristics by block group in 2000 and 2010 are presented in Table 2.1. Overall, the Massachusetts state population grew by 3.1% between 2000 and 2010. The state contained 81.9% Non-Hispanic white in 2000 and 76.1% in 2010. The Hispanic population increased by 47% from 2000 to 2010, growing from 6.7% of the population to 9.6%. The percent of the population with less than a high school education decreased 3.9 percentage points from 2000 to 2010. Average inflation-adjusted median household income among all Massachusetts block groups was essentially unchanged, although the 10^{th} percentile value decreased by $3,383 (9.8%) and the 95th percentile value increased by $4,407 (3.4%), indicating growing income inequality.

We observe distinct population distribution changes between block groups categorized as rural or urban. The size of the population grew by 5.3% in urban block groups and decreased by 4.1% in rural block groups. Overall, a higher percentage of racial/ethnic minorities live in urban than in rural block groups. Urban non-Hispanic whites experienced the greatest change of any population group between 2000 and 2010, decreasing by 6.2%. Educational attainment distributions are similar between rural and urban block groups in both 2000 and 2010, and both experienced a decrease in the population with less than a high school education. In general, median household incomes were higher in rural than in urban block groups, and the growing income inequality was more pronounced in urban areas.

Table 2.1. Massachusetts Census 2000, Census 2010 and ACS 2006-2010 demographic and geographic subpopulation characteristics

	Full State					Urban					Rural				
	2000		2010		% Change[b]	2000		2010		% Change[b]	2000		2010		% Change[b]
	#	%	#	%		#	%	#	%		#	%	#	%	
Race/Ethnicity															
Total	6349097		6547629			5337451		5620943		5.3	885599		849293		
Non-Hispanic White	5197124	81.9	4984800	76.1	-5.7	4248651	79.6	4126251	73.4	-6.2	836963	94.5	790098	93.0	-1.5
Non-Hispanic Black	314472	5.0	391693	6.0	1.0	301276	5.6	380308	6.8	1.1	9923	1.1	9625	1.1	0.0
Non-Hispanic Asian	237006	3.7	347495	5.3	1.6	223262	4.2	331479	5.9	1.7	10159	1.1	15010	1.8	0.6
Non-Hispanic Other	173155	2.7	195987	3.0	0.3	156707	2.9	178692	3.2	0.2	12936	1.5	14843	1.7	0.3
Hispanic	427340	6.7	627654	9.6	2.9	407555	7.6	604213	10.7	3.1	15618	1.8	19717	2.3	0.6
Educational Attainment															
Total	4273275		4382378			3598210		3745562		2.8	600501		583138		
Less than High School	651093	15.2	495822	11.3	-3.9	575286	16.0	448584	12.0	-4.0	64965	10.8	41550	7.1	-3.7
High School Graduate	1165489	27.3	1171725	26.7	-0.5	978895	27.2	996370	26.6	-0.6	163986	27.3	157267	27.0	-0.3
Post-Secondary	1038398	24.3	1036622	23.7	-0.6	859029	23.9	867966	23.2	-0.7	160918	26.8	153673	26.4	-0.4
Bachelors	834554	19.5	961563	21.9	2.4	695403	19.3	817500	21.8	2.5	126717	21.1	134937	23.1	2.0
Graduate	583741	13.7	716646	16.4	2.7	489597	13.6	615142	16.4	2.8	83915	14.0	95711	16.4	2.4
	Full State					Urban					Rural				
	Mean	SD	5th	95th		Mean	SD	5th	95th		Mean	SD	5th	95th	
Median Household Income															
Census 2000, 2010 inflation-adjusted $	70,169	32,225	27,152	127,964		69,110	32,221	26,535	126,745		80,098	31,413	42,402	142,350	
Census 2010, 2010 inflation-adjusted $	70,114	34,262	31,125	132,371		69,070	34,785	20,543	132,197		80,172	29,315	42,024	139,853	

[a]Includes all block groups classified as urbanized, urban cluster, or rural by the U.S. Census. Due to the small number of block groups categorized as "urban cluster," stratified results for this category are not presented.
[b]Change in percent of total population

Population Weighted Concentrations

Based on modelled $PM_{2.5}$ and NO_2, we find that average annual $PM_{2.5}$ concentrations across the state decreased by 35% between 2003 and 2010, and that average annual NO_2 concentrations decreased by 24% between 2005 and 2010. Concentrations were consistently lower in rural than urban areas, but patterns of change remained the same between the two strata (Figures 2.1a and 2.1b).

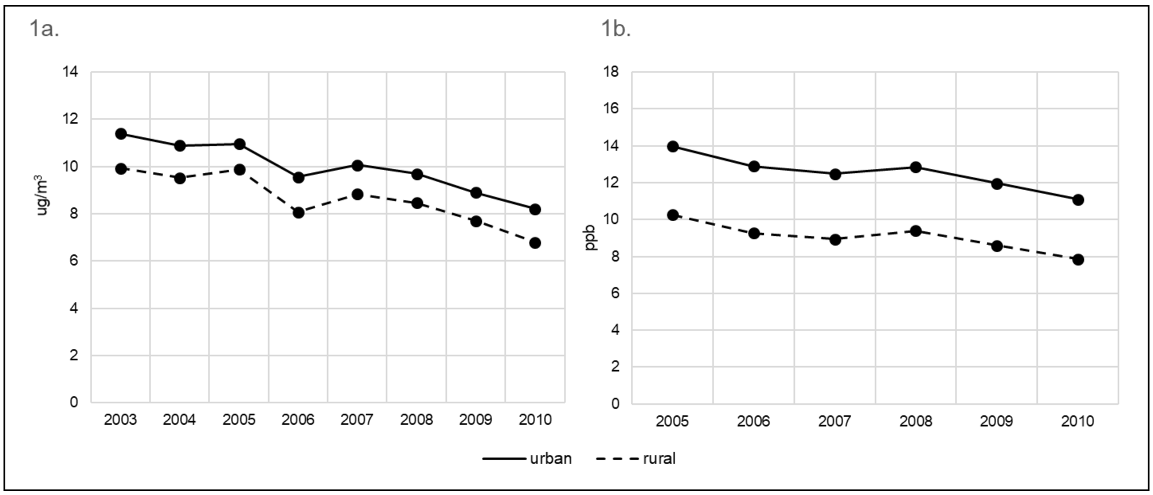


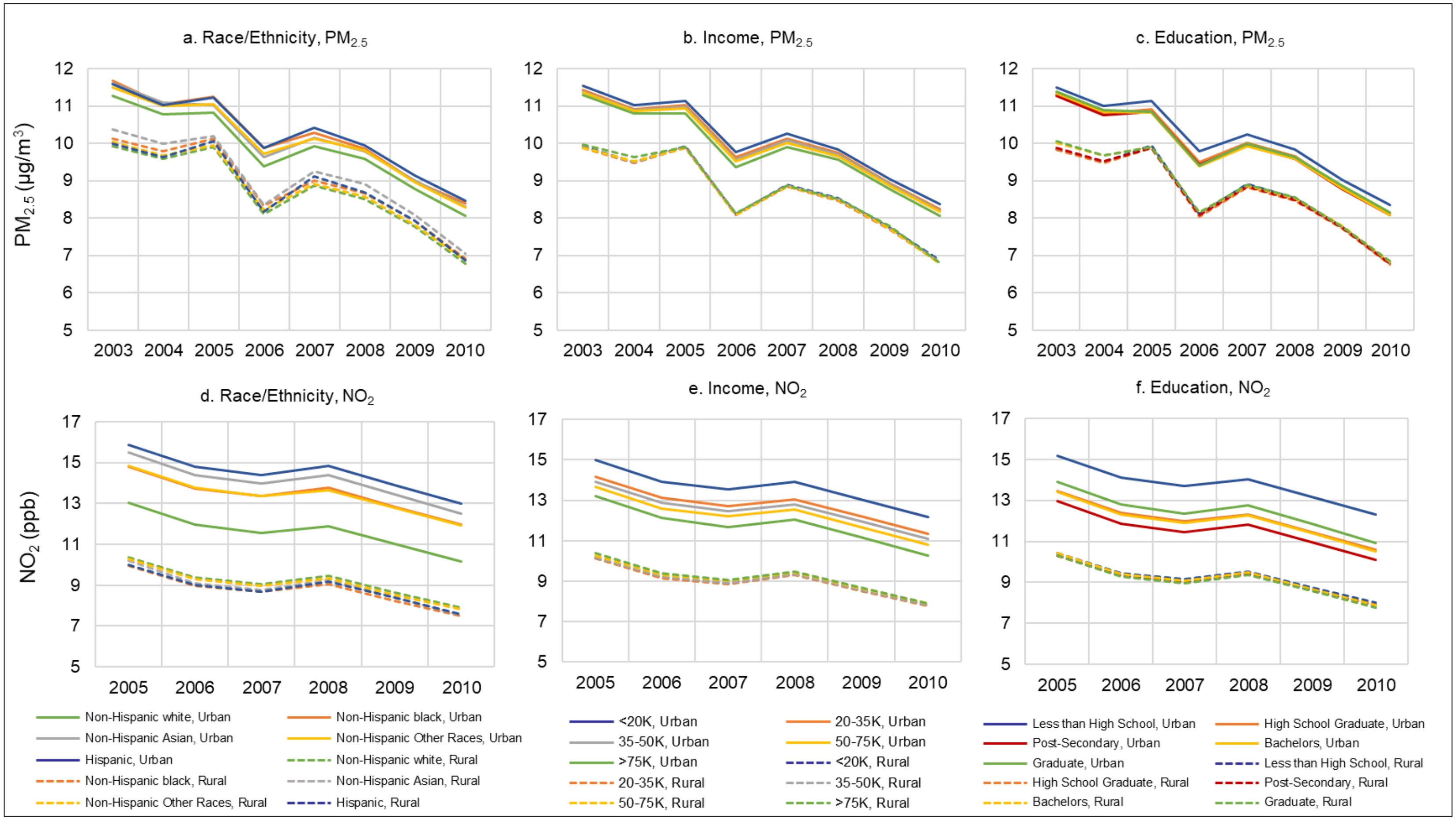
Figure 2.1a. Annual Average $PM_{2.5}$ Concentrations in Massachusetts, 2003-2010

Figure 2.1b. Annual Average NO_2 Concentrations in Massachusetts, 2005-2010

Tables 2.2 and 2.3 display snapshots of subgroup population-weighted $PM_{2.5}$ and NO_2 concentrations, along with absolute and relative (percent) change in exposure over the study period for the full state and rural/urban classifications. Population-weighted concentrations for $PM_{2.5}$ in 2003 and NO_2 concentrations in 2005 were calculated using the Census 2000 population, approximating the spatial and demographic distributions of the population in those years. For $PM_{2.5}$ and NO_2 population-weighted concentrations in 2010, the Census 2010 and ACS 2006-2010 populations were used to characterize the population distribution (Tables 2.2 and 2.3). Figures 2.2a-f display population weighted trends in $PM_{2.5}$ and NO_2 concentration for each year between 2003 and 2010 using demographic data from Census 2010 and ACS 2006-2010, allowing us to isolate the effects of changing concentrations from any sociodemographic shifts. Longitudinal changes in population-weighted concentrations using demographic data from Census 2000 can be found in the Appendix (Figures S2.1a-S2.1f).

$PM_{2.5}$. Overall, weighted $PM_{2.5}$ exposures in 2003 across the state ranged from 11.1 to 11.7 μg/m^3 across racial/ethnic groups, with ranges in urban block groups from 11.3 to 11.8 μg/m^3 and in rural block groups from 10.1 to 10.8 μg/m^3 (Table 2.2). In 2010, weighted $PM_{2.5}$ exposures ranged across racial/ethnic groups ranged from 7.8 to 8.4 μg/m^3, 8.1 to 8.5 μg/m^3 and 6.8 to 7.0 μg/m^3 across the state and among urban and rural populations, respectively. Across the state in 2003, $PM_{2.5}$ concentrations were highest for the non-Hispanic black (11.7 μg/m^3) population among racial/ethnic groups, those with less than a high school education (11.3 μg/m^3) among education groups, and those with incomes less than $20,000 per year (11.4 μg/m^3) among income groups. Among racial/ethnic groups in 2003, the greatest difference in population weighted concentration was between non-Hispanic whites (11.1 μg/m^3) and non-Hispanic blacks (11.7 μg/m^3), and in 2010 the greatest difference was between non-Hispanic whites (7.8 μg/m^3) and both Hispanic and non-Hispanic black (8.4 μg/m^3) populations. These patterns were present in urban but not rural block groups. The absolute decrease in $PM_{2.5}$ over time was relatively homogenous across all population groups.

Figures 2.2a-c display population weighted trends in $PM_{2.5}$ exposures from 2003 to 2010, holding Census 2010 and ACS 2006-2010 data constant. Among racial and ethnic groups, non-Hispanic Asian populations experienced the largest decrease in $PM_{2.5}$ exposures between 2003 and 2010 in both urban (28.8%) and rural (33.6%) locations, whereas the urban Hispanic population experienced the lowest percentage decrease (27.3%). Among rural income groups, the greatest decrease in $PM_{2.5}$ was observed for median incomes above $75,000 per year (33.5%), a pattern that differed from urban income groups. In general, the population groups with the highest exposures in 2003 experienced lower relative decreases in exposures over time, consistent with similar absolute reductions across populations. Holding the Census 2000 population constant over annual $PM_{2.5}$ concentrations reveals similar results (Figures S2.2a-S2.2c).



Figures 2.2a-2.2f. Population-Weighted Annual Average $PM_{2.5}$ (a-c) and NO_2 (d-f) concentrations by Census 2010 and ACS 2006-2010 Demographic and Geographic Subpopulations

Table 2.2 Population-weighted annual average $PM_{2.5}$ ($\mu g/m^3$) Concentrations by Census 2000, Census 2010 and ACS 2006-2010 demographic and geographic subpopulations

	Full State				Urban				Rural			
	$PM_{2.5}$ 2003, Census 2000	$PM_{2.5}$ 2010, Census 2010	Absolute Change[a]	Relative Change[b]	$PM_{2.5}$ 2003, Census 2000	$PM_{2.5}$ 2010, Census 2010	Absolute Change[a]	Relative Change[b]	$PM_{2.5}$ 2003, Census 2000	$PM_{2.5}$ 2010, Census 2010	Absolute Change[a]	Relative Change[b]
Race/Ethnicity												
Total	11.2	8.0	-3.2	-28.7	11.4	8.1	-3.2	-28.3	10.1	6.8	-3.3	-32.7
non-Hispanic white	11.1	7.8	-3.2	-29.2	11.3	8.1	-3.2	-28.6	10.1	6.8	-3.3	-32.7
non-Hispanic black	11.7	8.4	-3.4	-28.6	11.8	8.4	-3.4	-28.6	10.4	6.9	-3.5	-33.6
non-Hispanic Asian	11.6	8.2	-3.4	-29.0	11.7	8.3	-3.4	-28.8	10.8	7.0	-3.8	-35.0
non-Hispanic other	11.4	8.2	-3.3	-28.6	11.6	8.3	-3.3	-28.4	10.2	6.9	-3.3	-32.6
Hispanic	11.6	8.4	-3.2	-27.4	11.6	8.5	-3.2	-27.3	10.5	6.9	-3.6	-34.4
	$PM_{2.5}$ 2003 Census 2000	$PM_{2.5}$ 2010, ACS 2010	Absolute Change[a]	Relative Change[b]	$PM_{2.5}$ 2003, Census 2000	$PM_{2.5}$ 2010 ACS 2010	Absolute Change[a]	Relative Change[b]	$PM_{2.5}$ 2003, Census 2000	$PM_{2.5}$ 2010, ACS 2010	Absolute Change[a]	Relative Change[b]
Educational Attainment												
Total	11.2	7.9	-3.2	-28.8	11.4	8.1	-3.2	-28.4	10.1	6.8	-3.3	-32.8
< High School	11.3	8.2	-3.1	-27.6	11.5	8.3	-3.1	-27.3	10.1	6.8	-3.4	-33.2
High School Grad	11.1	7.9	-3.2	-28.7	11.3	8.1	-3.2	-28.2	10.0	6.8	-3.3	-32.7
Post-Secondary	11.1	7.9	-3.2	-28.9	11.3	8.1	-3.2	-28.5	10.0	6.8	-3.3	-32.5
Bachelors	11.2	7.9	-3.3	-29.3	11.3	8.1	-3.3	-28.8	10.2	6.8	-3.4	-33.1
Masters	11.2	8.0	-3.3	-29.0	11.4	8.2	-3.3	-28.6	10.2	6.8	-3.4	-33.0
Median Household Income												
Total	11.2	8.0	-3.2	-28.7	11.4	8.2	-3.2	-28.3	10.1	6.8	-3.3	-32.6
<20,000	11.4	8.2	-3.1	-27.6	11.5	8.4	-3.2	-27.4	10.2	6.9	-3.3	-32.4
20-35,000	11.2	8.1	-3.2	-28.1	11.4	8.2	-3.2	-27.8	10.0	6.8	-3.2	-32.0
35-50,000	11.2	8.0	-3.2	-28.3	11.4	8.2	-3.2	-28.0	10.0	6.8	-3.2	-31.7
50-75,000	11.1	8.0	-3.2	-28.3	11.3	8.2	-3.2	-28.1	10.0	6.8	-3.2	-32.1
>75,000	11.1	7.9	-3.3	-29.3	11.3	8.1	-3.2	-28.7	10.2	6.8	-3.4	-33.5

[a]Absolute change in concentrations ($\mu g/m^3$)
[b]Percentage change in concentrations

Table 2.3. Population-weighted annual average NO_2 (ppb) Concentrations by Census 2000, Census 2010 and ACS 2006-2010 demographic and geographic subpopulations

	Full State				Urban				Rural			
	NO_2 2005, Census 2000	NO_2 2010, Census 2010	Absolute Change[a]	Relative Change[b]	NO_2 2005, Census 2000	NO_2 2010, Census 2010	Absolute Change[a]	Relative Change[b]	NO_2 2005, Census 2000	NO_2 2010, Census 2010	Absolute Change[a]	Relative Change[b]
Race/Ethnicity												
Total	13.2	10.4	-2.8	-21.4	13.7	10.8	-2.9	-21.2	10.5	7.9	-2.6	-24.8
non-Hispanic white	12.7	9.8	-2.9	-23.0	13.0	10.2	-2.9	-22.2	10.4	7.9	-2.5	-24.4
non-Hispanic black	14.9	11.8	-3.1	-20.8	14.8	12.0	-2.9	-19.3	10.9	7.5	-3.3	-30.8
non-Hispanic Asian	15.8	12.3	-3.5	-22.2	15.5	12.5	-3.0	-19.2	11.6	7.5	-4.0	-35.0
non-Hispanic other	14.9	11.5	-3.3	-22.5	14.8	11.9	-2.9	-19.6	10.6	7.8	-2.8	-26.3
Hispanic	15.8	12.8	-3.0	-19.2	15.9	13.0	-2.9	-18.0	11.2	7.6	-3.6	-32.0
	NO_2 2005 Census 2000	NO_2 2010, ACS 2010	Absolute Change[a]	Relative Change[b]	NO_2 2005, Census 2000	NO_2 2010, ACS 2010	Absolute Change[a]	Relative Change[b]	NO_2 2005, Census 2000	NO_2 2010, ACS 2010	Absolute Change[a]	Relative Change[b]
Educational Attainment												
Total	13.1	10.3	-2.8	-21.7	13.7	10.7	-3.0	-21.7	10.4	7.9	-2.6	-24.5
< High School	14.5	11.9	-2.7	-18.3	15.1	12.3	-2.8	-18.5	10.6	8.0	-2.6	-24.4
High School Grad	13.0	10.2	-2.8	-21.5	13.5	10.6	-2.9	-21.5	10.4	7.9	-2.5	-23.8
Post-Secondary	12.5	9.7	-2.8	-22.4	13.0	10.1	-2.9	-22.4	10.4	7.9	-2.5	-24.0
Bachelors	12.9	10.1	-2.9	-22.0	13.5	10.5	-3.0	-22.1	10.5	7.9	-2.6	-24.7
Masters	13.3	10.5	-2.8	-21.2	13.9	10.9	-2.9	-21.1	10.5	7.8	-2.8	-26.2
Total	13.3	10.5	-2.8	-21.4	13.9	10.9	-3.0	-21.4	10.4	7.9	-2.6	-24.5
Median Household Income												
<20,000	14.4	11.7	-2.7	-18.5	15.0	12.2	-2.8	-18.8	10.5	7.8	-2.6	-24.4
20-35,000	13.6	10.9	-2.7	-20.1	14.2	11.3	-2.9	-20.1	10.3	7.8	-2.5	-23.8
35-50,000	13.4	10.6	-2.7	-20.3	13.9	11.1	-2.8	-20.4	10.3	7.8	-2.5	-24.0
50-75,000	13.0	10.4	-2.6	-20.2	13.6	10.8	-2.8	-20.3	10.4	7.9	-2.6	-24.7
>75,000	12.7	9.9	-2.8	-22.1	13.2	10.3	-2.9	-21.9	10.6	7.9	-2.8	-26.2

[a]Absolute change in concentrations (ppb)
[b]Percentage change in concentrations

NO_2. Table 2.3 displays population weighted NO_2 concentrations, absolute and percent decrease in exposure for the full state, and patterns of exposure stratified by sociodemographic characteristics and rural/urban status. Across the state in both 2005 and 2010, NO_2 concentrations were highest for Hispanic populations (15.8 ppb in 2005 and 12.8 ppb in 2010), those with less than a high school education (14.5 ppb in 2005 and 11.9 ppb in 2010) and households in the lowest income bracket (14.4 ppb in 2005 and 11.7 ppb in 2010). Patterns were identical in urban block groups. However, in rural block groups, the non-Hispanic Asian population experienced the highest exposure in 2005 (11.6 ppb), while non-Hispanic whites (7.9 ppb) had the highest NO_2 burden in 2010. Those in the highest income bracket in rural block groups also experienced the highest NO_2 concentrations in both 2005 and 2010.

Figures 2d-f display trends in population-weighted NO_2 concentrations from 2005 to 2010 using demographic data from Census 2010 and ACS 2006-2010. In general, the rate of NO_2 concentration decrease was greater in rural than urban block groups. Similar to $PM_{2.5}$, population-weighted NO_2 exposure inequality existed for urban, but not rural, sociodemographic groups. Trends in population-weighted NO_2 exposure make clear that patterns of inequalities persisted from 2005 to 2010, and that urban racial/ethnic minorities, low income and education groups remained the highest exposure groups. Results are similar when applied to the Census 2000 population (Figures S2.1d-S2.1f)

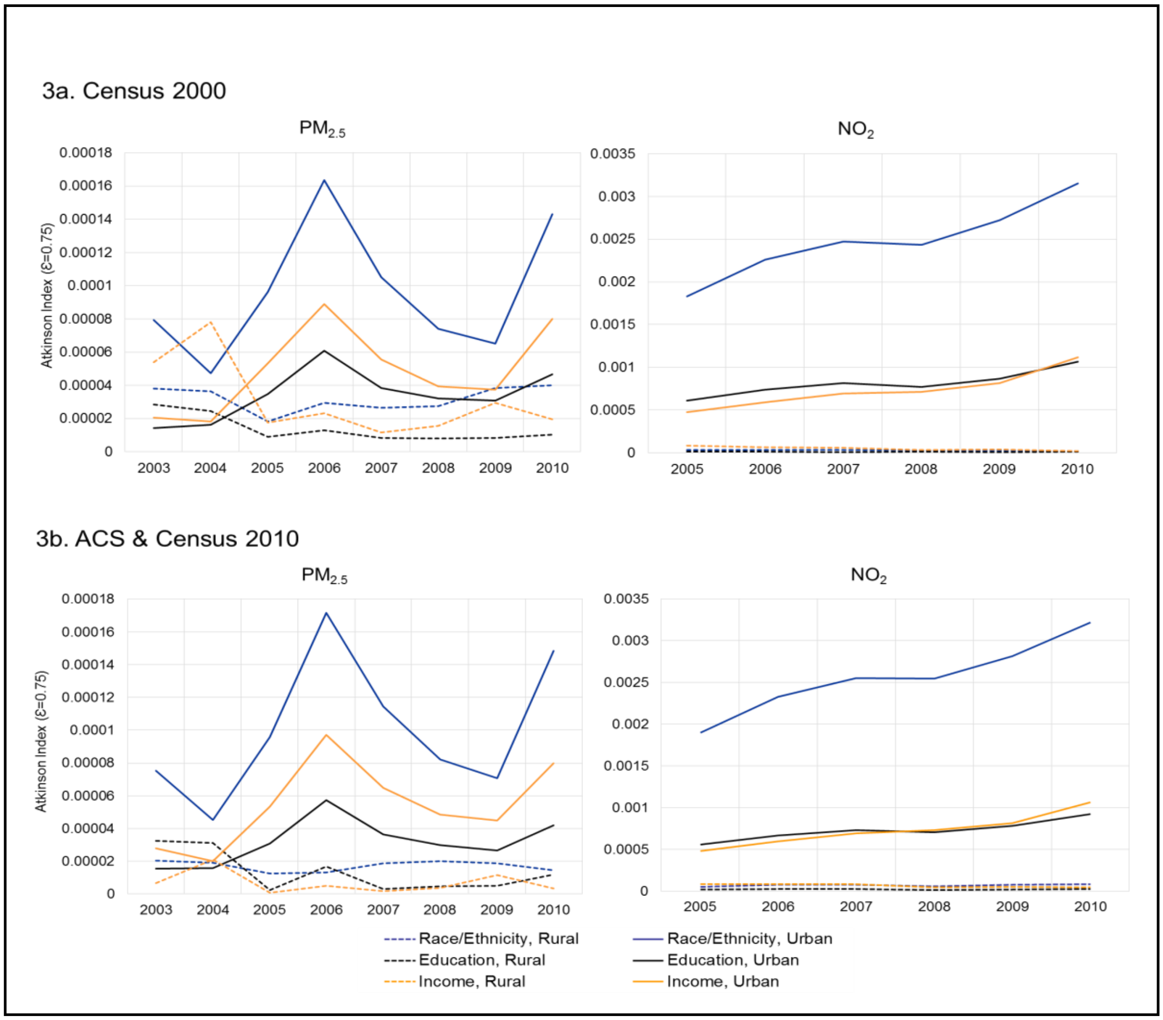


Figure 2.3. Between-Group Inequality in Population-Weighted Annual Average $PM_{2.5}$ and NO_2 Concentrations.

Figure 2.3a displays results for $PM_{2.5}$ and NO_2 inequality for the Census 2000 Demographic and Geographic Subpopulations.

Figure 2.3b displays results for $PM_{2.5}$ and NO_2 inequality for the Census 2010 and ACS 2006-2010 Demographic and Geographic Subpopulations.

Atkinson Index

$PM_{2.5}$. We estimate the AI for each year, separately using Census 2000 and Census 2010/ACS 2006-2010, to determine how exposure inequality has evolved over time and whether this is related to concentration patterns or changing demographics.

Overall, AI trends and values are relatively insensitive to the choice of population data (Figure 2.3). The principal difference between the two demographic years is demonstrated in modestly higher rural exposure inequality trends among racial/ethnic and income groups for the Census 2000 compared to the Census 2010 population. These results indicate that both population mobility and shifting $PM_{2.5}$ distributions contribute to rural exposure inequality trends. Exposure inequality trends among all subgroups living in urban block groups are nearly identical between 2000 and 2010, indicating that shifting $PM_{2.5}$ distributions (and not population mobility) are likely driving observed exposure inequality trends in urban areas.

Between-sociodemographic group $PM_{2.5}$ inequality using the AI reveals peaks of increased and decreased inequality over time as a result of $PM_{2.5}$ concentration distributions in urban block groups, and a slight decreasing trend among rural block groups (Figure 2.3a). We additionally find that inequality is generally greater in magnitude, especially after 2005, in urban block groups. Although the AI values are generally low, they are consistently higher for racial/ethnic groups compared to inequality among income and education groups. The AI results seen here are explained by non-Hispanic black and Hispanic populations, low-income, and low educational attainment populations consistently experiencing a greater $PM_{2.5}$ burden than the other racial/ethnic, income and education groups (Table 2.2). As all sociodemographic groups experience a similar absolute decrease in exposure, the lowest exposed groups, such as non-Hispanic whites, undergo greater relative rates of exposure decline over time.

NO_2. We observe distinctly different patterns in quantified NO_2 inequality as

compared to $PM_{2.5}$ inequality (Figure 2.3). AI values are generally low but approximately one order of magnitude greater for NO_2 compared to $PM_{2.5}$. Between 2005 and 2010 there is a slight increase in NO_2 inequality among all population strata located in urban block groups. Inequality was greatest for racial/ethnic subpopulations in urban block groups, which also experienced the greatest rate of increase in AI over time. AI results were similar when using Census 2000 and Census 2010/ACS 2006-2010 population data.

Discussion

Our study builds on previous environmental inequality analyses that use measures of proximity or coarsely-resolved measures of air pollution exposure and investigate inequality at one point in time by incorporating longitudinal Census data and pollution concentrations. This work additionally builds on the current literature by employing a novel application of the AI to formally quantify inequality between population groups over time.

Although modeled and monitored air pollution data have demonstrated longitudinal reductions in concentrations, our highly-resolved and stratified analyses provided some novel insights with respect to exposure inequalities. For example, we found distinct differences in population-weighted concentration patterns and trends over time between $PM_{2.5}$ and NO_2 and between urban and rural geographic areas. Urban areas contain greater densities of low-income, non-white and low-educational attainment populations and $PM_{2.5}$ and NO_2 pollution sources, contributing to some exposure heterogeneity and potential inequalities. Greater concentration of urban air pollution sources is reflected in our findings of non-Hispanic blacks, individuals with lower educational attainment, and households with an annual income of <$20K as the most burdened population groups for both NO_2 and $PM_{2.5}$ concentrations throughout the state.

That said, $PM_{2.5}$ concentrations are more regional than local in nature because they are derived from a wide variety of sources, with a strong contribution from secondary pollutant formation and long-range transport (Zheng et al., 2002). NO_2 is strongly linked to automobiles and other mobile sources and tends to exhibit high intra-urban variability. As such, it has greater potential for exposure inequalities in urban settings. $PM_{2.5}$ is a regionally-based pollutant, exhibiting less spatial variability in urban areas, leading to smaller exposure disparities compared to NO_2 (Clougherty et al., 2008). These pollutant-specific characteristics are reflected in our finding of NO_2 inequality that is greater in magnitude than $PM_{2.5}$ inequality. Higher NO_2 inequality growth rates within urban areas, especially between racial/ethnic groups, may further be explained by increased local source emissions, such as higher traffic counts over time or increased transportation infrastructure in Boston neighborhoods containing high proportions of non-Hispanic black and Hispanic populations (Brugge et al., 2015; Levy et al., 2001).

In rural settings, NO_2 exposures were disproportionately higher for Hispanic populations, individuals with lower educational attainment, and households with lower income in urban block groups, but higher for non-Hispanic Asians and the wealthiest population groups in rural block groups. This could reflect the fact that roadway proximity tends to decrease property value in urban areas, but may potentially increase them in rural areas (Bateman et al., 2001; Lake et al., 1998). Analyses that did not stratify by urban/rural status would not appropriately characterize exposure inequality or capture key between-group differences.

We additionally found fluctuations in $PM_{2.5}$ inequality and increasing trends in between-group NO_2 inequality, despite similar absolute rates of $PM_{2.5}$ and NO_2 decline across population groups. Uniform absolute reductions are beneficial to all but do not decrease exposure inequalities, and in fact, tend to increase them for metrics such as the AI given growing relative differences. Similar AI trends using Census 2000 and Census 2010 populations indicates that sociodemographic mobility is not the main driver of urban $PM_{2.5}$ and statewide NO_2 inequality trends, although it could remain a contributing factor. It would be informative for future studies to examine inequality trends using annual demographic data where available, holding ambient concentrations constant.

Because most environmental inequality studies rely on cross-sectional data, they do not inform our understanding of the components that contribute to changing inequality over time (Bell and Ebisu, 2012; Lopez, 2002; Miranda et al., 2011; Pastor et al., 2004; Rosofsky et al., 2014). Our application of the AI to characterize exposure inequality addresses this literature gap by separately examining population and air pollution patterns, to determine which best explains changing inequality. To our knowledge, only one study to date has applied the AI to describe spatial patterns of pollution across sociodemographic characteristics and between rural and urban areas (Clark et al., 2014). Clark et al. (2014) findings of population-weighted racial/ethnic and income disparity for NO_2 exposure nationwide were similar to our results: nonwhite and low-income populations experienced the greatest burden of NO_2 exposure, and these disparities were more pronounced in large urban areas than rural areas.

A handful of studies within the environmental inequality literature have also moved to address this gap (Kravitz-Wirtz et al., 2016; Mohai and Saha, 2015a; Pastor et al., 2001). One recent study by Kravitz-Wirtz et al. (2016) examined trends in racial and ethnic disparities in exposure to neighborhood air pollution across the U.S., while controlling for individual and neighborhood-level changes over time. The authors found that black and Hispanic participants were disproportionately exposed to higher concentrations of NO_2, $PM_{2.5}$ and PM_{10} compared to white participants, and that concentrations decreased for all racial and ethnic groups over time. In contrast to our findings, rate of decline in $PM_{2.5}$ and NO_2 exposure among black and Hispanic participants were more pronounced than for white participants. The authors hypothesized that these findings are explained by more rapid decreases in pollution in urban areas, where black and Hispanic participants of the study reside. However, our study found the opposite effect, with concentrations of $PM_{2.5}$ and NO_2 falling more rapidly in rural areas, and for the non-Hispanic white population.

In general, our AI values are quite small, and we observed small absolute differences in $PM_{2.5}$ and NO_2 concentrations between population groups. However, AI values cannot be reasonably compared across contexts (i.e., income spans many orders of magnitude, whereas ambient air pollution has a narrower range within a state), and are most meaningful for comparisons over time or between pollutants analyzed similarly. In addition, the exposure differences may be large enough to contribute to health disparities (Atkinson et al., 2014; Brauer et al., 2008; Clark et al., 2014; Shi et al., 2016). For instance, in a recent study by Shi et al. (2016), all-cause mortality increased by 0.9% per

$\mu g/m^3$ increase in long-term $PM_{2.5}$ concentrations even when restricted to ambient concentrations below 10 $\mu g/m^3$. The 0.6 $\mu g/m^3$ difference in exposure in urban areas in 2010 for non-Hispanic whites versus Hispanics would therefore translate into a 0.5% increase in mortality rates, all else being equal. Further, the population subgroups found to have the highest population-weighted $PM_{2.5}$ and NO_2 concentrations also tend to have higher baseline rates of asthma and cardiovascular disease, leaving them more vulnerable to persistent, longitudinal air pollution exposure (Crain et al., 1994; Jones et al., 2009; O'Neill et al., 2003).

Our findings demonstrating inequitable pollution exposure by SES and race/ethnicity are supported by evidence of environmental inequality that is firmly established in the academic literature (Lopez, 2002; Mohai and Bryant, 1992; Mohai and Saha, 2015b; Morello-Frosch and Lopez, 2006). Further, a growing number of studies have demonstrated environmental inequality specific to $PM_{2.5}$ and NO_2 in Massachusetts and nationwide using Census data (Clark et al., 2014; Miranda et al., 2011; Yanosky et al., 2008). For instance, Miranda et al. (2011) used an air quality ranking approach to assess environmental justice dimensions of air pollution exposure, finding that the proportion of non-Hispanic black residents in the 20% of counties in the United States with the poorest air quality was twice that in those counties with the most favorable air quality. Yanosky et al. (2008) evaluated whether predicted NO_2 concentrations are associated with socioeconomic position, after controlling for spatial autocorrelation in Worcester, Massachusetts. They found that block group NO_2 concentrations exhibit a significant negative association with median household income, and that rates of poverty and low educational attainment populations rose by 3.1% and 3.4%, respectively, with every one standard deviation increase in block group mean NO_2.

Despite employing novel inequality-based methods using data of high temporal and geographic resolution, there are some limitations that merit discussion. The use of Census data restricts our ability to examine disparities at the individual/household level. Using personal monitors is not feasible at this scale, so we assigned modeled $PM_{2.5}$ and NO_2 concentrations to each block group to approximate individual exposure, thereby limiting potential variability in exposure across the population. Our results consequently do not incorporate individual mobility or characteristics that may provide a more comprehensive understanding of the drivers of inequality. However, the inputs used to assign block-group level exposures are advantageous over proximity-based and aggregation methods that ignore chemical fate and transport and local meteorological conditions (Chakraborty et al., 2011; Lucier et al., 2011; Mohai et al., 2011; Pastor et al., 2001). These predictions are also an improvement over EI studies that use predicted concentrations over coarse geographic and temporal resolutions (Hajat et al., 2015; Kravitz-Wirtz et al., 2016; Mohai and Saha, 2015b; Morello-Frosch and Jesdale, 2006; Pope et al., 2016). A 1 km^2 resolution is adequate for regionally-based pollutants, such as $PM_{2.5}$, but may ignore local hotspots for locally-based pollutants such as NO_2. Conversely, smaller geographic resolutions may introduce bias related to individual mobility (Setton et al., 2011)

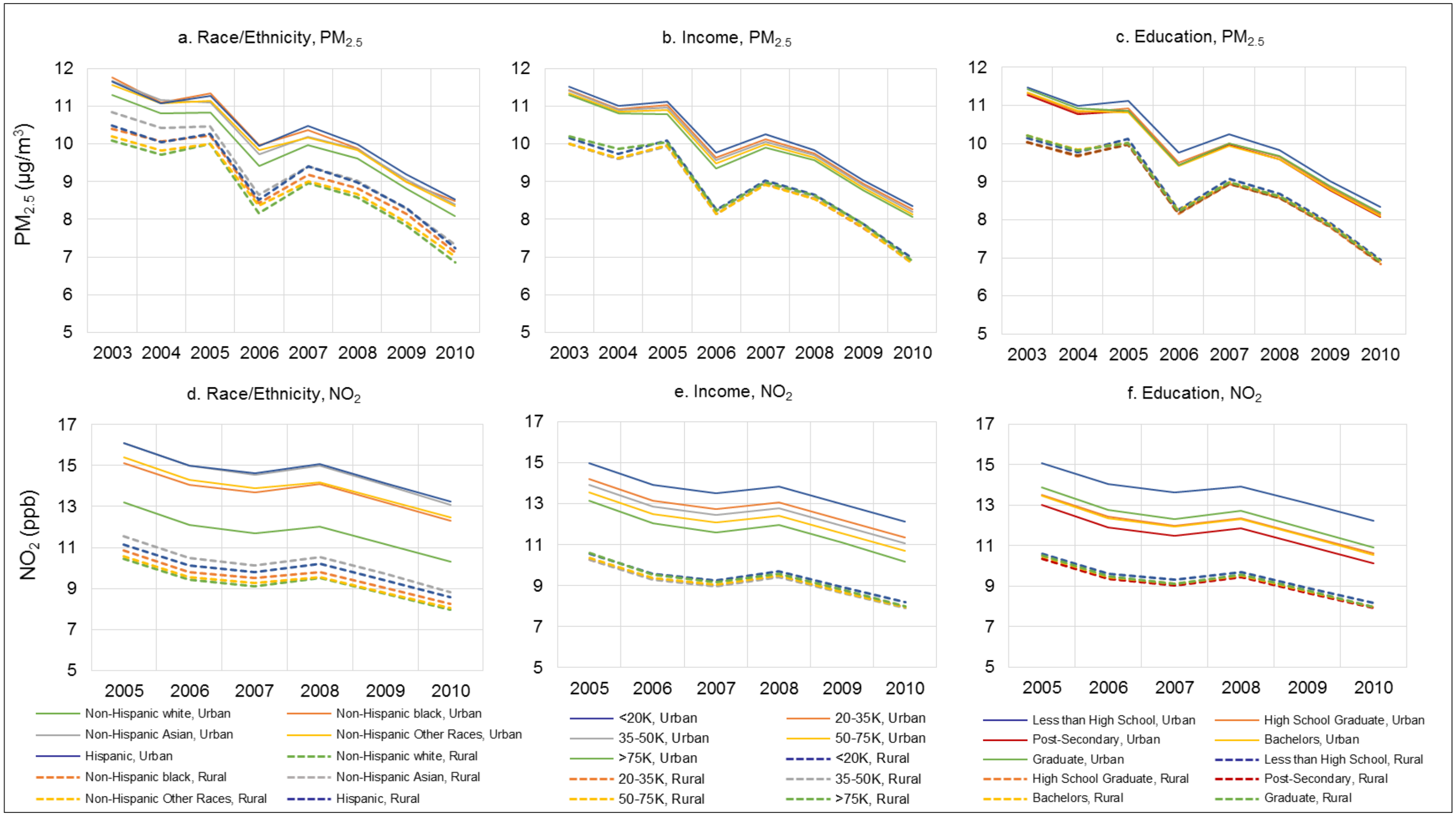
We acknowledge that the temporal misalignment of $PM_{2.5}$ (years 2003-2010) and NO_2 (years 2005-2010) with Census data for the year 2000 or 2010 prevents a precise characterization of exposure patterns over time. However, as discussed above, the relative stability of our inequality measures across different population data indicates that this is a minor source of error.

As a final overall limitation, we only studied inequalities in outdoor ambient air pollution exposures; low socioeconomic status groups may be disproportionately exposed to indoor-generated exposures or from indoor exposure to outdoor pollutants due to older, leakier housing stock (Adamkiewicz et al., 2011). Taking into account the full exposure profile of both indoor and outdoor-generated air pollutants may reveal a more striking characterization of exposure inequality between population groups.

Conclusion

Despite overall reductions in ambient air pollution concentrations and decreased industrialization, we found that air pollution inequalities have slightly increased over time when measured on a relative scale, and that group-specific concentrations are most disparate between racial/ethnic groups. Greater inequalities in urban areas, where there is often substantial segregation, reinforces the importance of targeted exposure reduction strategies within vulnerable populations and neighborhoods. Ultimately, there is a complex dynamic wherein changing sociodemographics over time may impact land use decisions, enforcement policy measures, and other factors influencing emissions. To complement these findings, more studies that utilize longitudinal, individual-level data are needed to understand population mobility and individual factors that affect exposure disparities.

Supplement



Figures S2.1a-S2.1f. Population-Weighted Annual Average $PM_{2.5}$ (a-c) and NO_2 (d-f) Concentrations by Census 2000 Demographic and Geographic Subpopulations

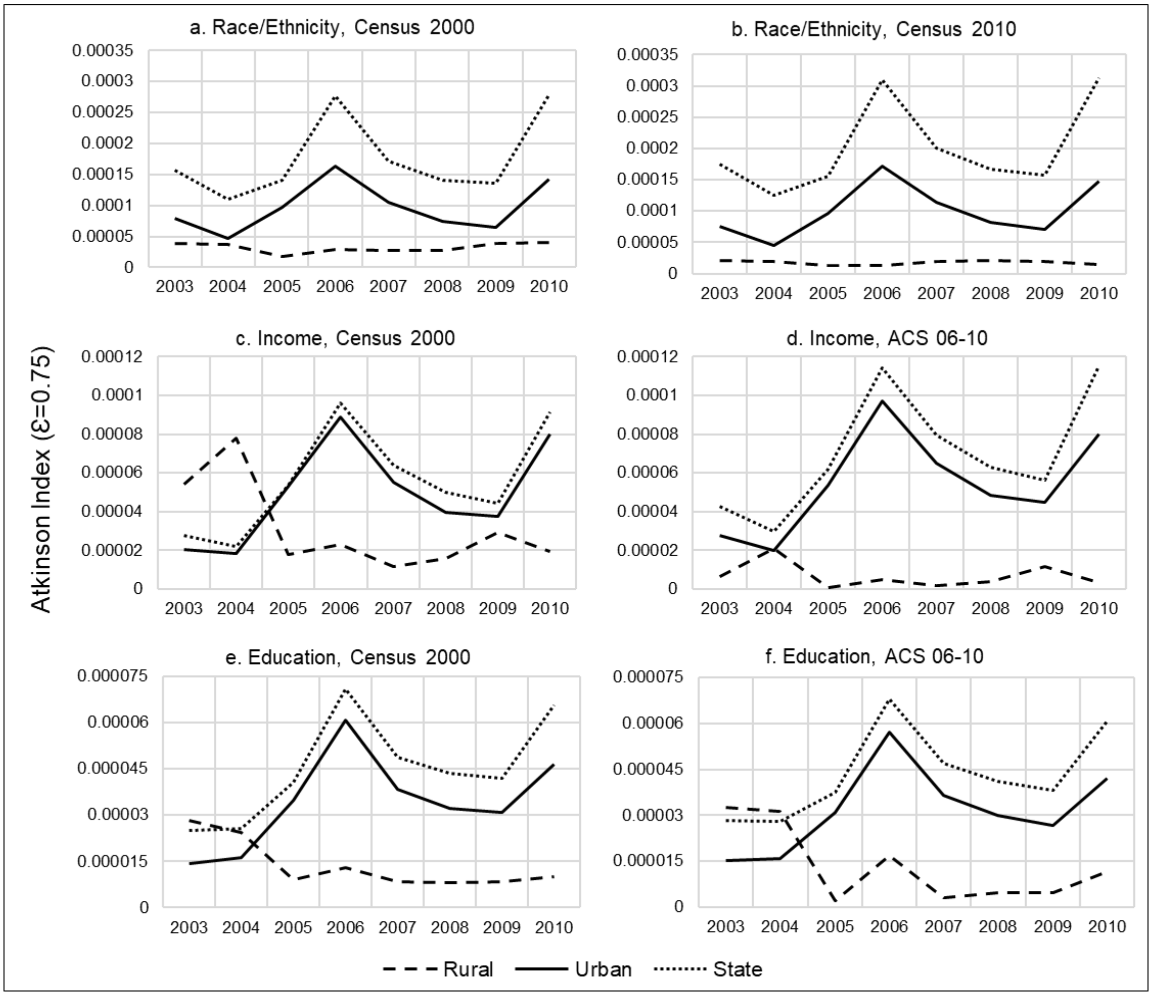


Figure S2.2. Between-Group Inequality in Population-Weighted Annual Average $PM_{2.5}$ Concentrations by Census 2000, 2010 and ACS 2006-2010 Demographic and Geographic Subpopulations

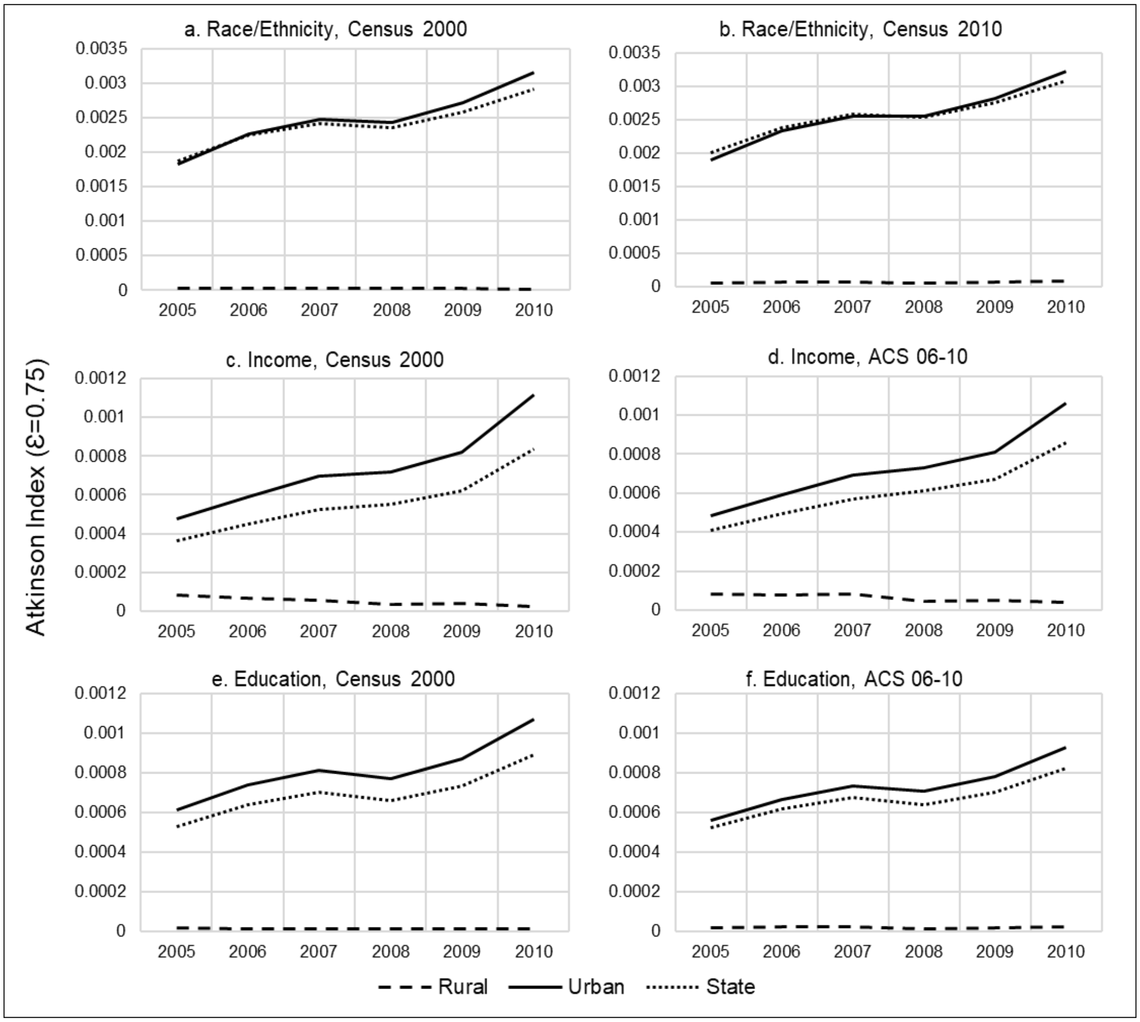


Figure S2.3. Between-Group Inequality in Population-Weighted Annual Average NO_2 Concentrations by Census 2000, 2010 and ACS 2006-2010 Demographic and Geographic Subpopulations

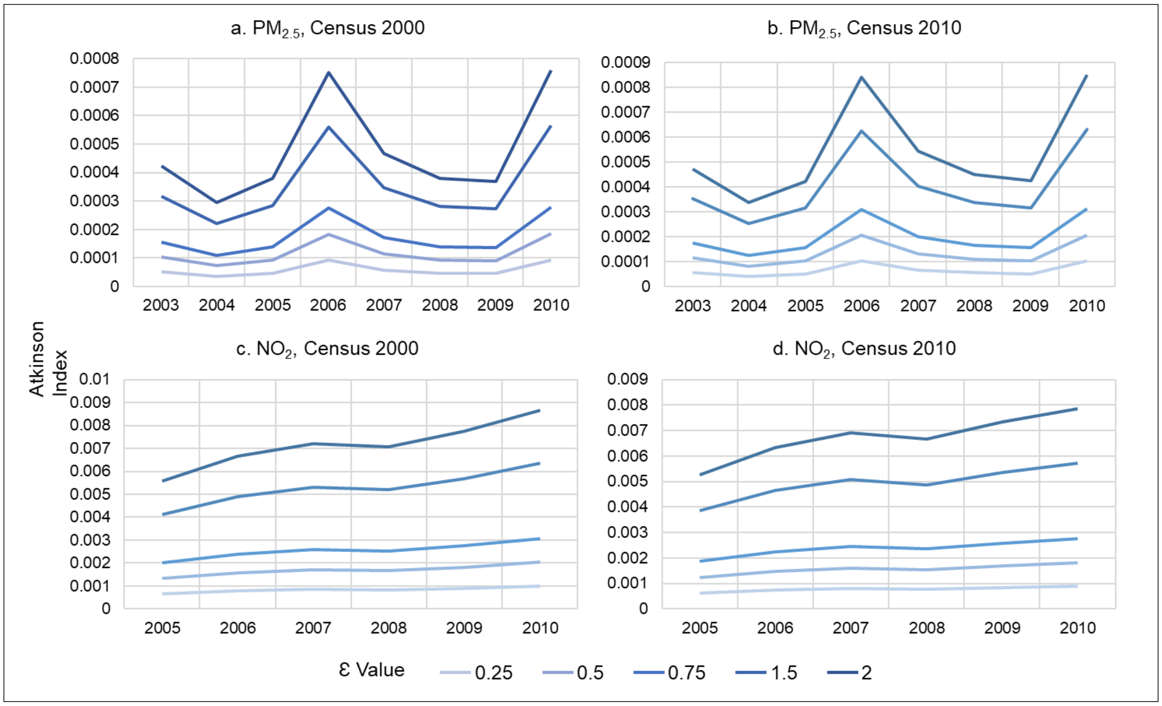


Figure S2.4. Between-Group Atkinson Index Values for Population-Weighted Annual Average $PM_{2.5}$ and NO_2 Concentrations by Race/Ethnicity for all Massachusetts Block Groups, Across Alternative Inequality Aversion Parameters

CHAPTER 3. THE IMPACT OF AIR EXCHANGE RATE ON AMBIENT AIR POLLUTION EXPOSURE AND INEQUALITIES ACROSS ALL RESIDENTIAL PARCELS IN MASSACHUSETTS

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Abstract

Individual housing characteristics can modify outdoor ambient air pollution infiltration through air exchange rate (AER). Due to time and labor-intensive methods needed to measure AER, few studies have characterized AER distributions across large geographic areas. Using publicly available data and combined physical and empirical models associating AER with housing characteristics, we estimated AER for all Massachusetts residential parcels. We then conducted an exposure disparities analysis, considering ambient $PM_{2.5}$ concentrations and residential AERs. Median AERs (h^{-1}) for winter and summer were 0.74 (IQR: 0.47-1.09) and 0.36 (IQR: 0.23-0.57) with closed windows, respectively, with lower AERs for single family homes. Across Massachusetts residential parcels, variability of indoor concentrations of ambient origin was twice that of ambient concentrations. Housing parcels above the 90th percentile of both AER and ambient $PM_{2.5}$ (i.e. the leakiest homes in areas of highest ambient air pollution) – versus the 10th percentile – were located in neighborhoods with higher proportions of Hispanics (20.0% vs 2.0%), households with an annual income of less than $20,000 (26.0% vs. 7.5%) and individuals with less than a high school degree (23.2% vs. 5.8%). Our approach can be applied in epidemiological studies to estimate exposure modifiers or to characterize exposure disparities that are not solely based on ambient concentrations.

Background

Exposure to ambient fine particulate matter ($PM_{2.5}$) contributes significantly to the global disease burden, with health impacts that include short gestational age and low birth weight related to prenatal exposures, negative cognitive and cardiovascular outcomes, respiratory illnesses, and all-cause mortality associated with postnatal exposures (Anderson et al., 2013; Atkinson et al., 2014; Volk et al., 2013b; Zheng et al., 2016; Zhu et al., 2015). Most epidemiological studies of air pollution rely on ambient concentrations as a surrogate for personal exposure (Brauer et al., 2008; Shi et al., 2016; Zhu et al., 2015), ignoring exposure variability that can occur due to individuals spending time in multiple built environments, particularly their home. In the United States, individuals spend approximately 87% of their time indoors and 69% of the time in their homes, which emphasizes the important role residential characteristics can play in modifying individual exposure to $PM_{2.5}$ of ambient origin (Klepeis et al., 2001). Personal exposure monitors have been used in epidemiological studies to capture exposure variability modified by the residential environment, but this method is costly and cannot be implemented on a large scale (Meng et al., 2005; Smargiassi et al., 2014). Thus, there is a need for straightforward methods to refine characterization of ambient air pollutant exposure on a large population-scale that can be used in health studies.

One factor that influences outdoor ambient air pollution infiltration into the home environment is air exchange rate (AER). However, there are challenges in characterizing AER over a large population. While measuring AER directly or modeling AER using detailed surveys may be feasible for smaller studies (Wallace et al., 2002; Yamamoto et al., 2010; Zota et al., 2005), estimating AER requires detailed, individual-level information about the building structure, the surrounding terrain, and meteorological conditions that impact air exchange, which are challenging to ascertain for large study populations and geographic extent. As a result of these limitations, few studies have examined how residential characteristics may modify ambient air pollution infiltration into homes using easily-accessible data, or characterized patterns of infiltration over large geographic areas (Baxter et al., 2013b; Chan et al., 2013; Shi et al., 2017; Taylor et al., 2016; Yamamoto et al., 2010).

Physical, empirical and mixed-methods models have been increasingly adopted to estimate AER in population-scale studies (Baxter et al., 2016, 2013a, Breen et al., 2015, 2010, Chan et al., 2013, 2005; Sarnat et al., 2013; Taylor et al., 2016; Zota et al., 2005). One study that used publicly-available data to estimate how geographic and temporal variations in residential AER modify the association between ambient air quality and health outcomes found that, at the zip code level, daily variability in average AER across a zip code explained heterogeneity in longitudinal asthma emergency department visits (Sarnat et al., 2013). A separate study used building simulation software to model how different housing types modify the indoor concentration of outdoor ambient $PM_{2.5}$, and mapped the results across dwellings in London (Taylor et al., 2016). However, these studies had limitations - the first estimated AER with coarse geographic resolution, not at residence level, and the second relied on building simulation software, which is both time consuming and requires specific expertise to apply.

Housing characteristics that modify ambient air pollution exposures have the potential for widening or narrowing the inequality gap in ambient exposures. Housing geography is closely linked to both residential segregation and poverty (Rauh et al., 2008), which are correlated with individual physical housing characteristics that can influence residential pollution and ultimately disease burden. For instance, low-socioeconomic status (SES) residents often live in smaller and older units, resulting in different household-level ventilation patterns (Adamkiewicz et al., 2011). Inadequate city code enforcement and residential instability may lead to deterioration in the neighborhood housing stock and value. Further, these same communities suffer a greater burden of outdoor ambient air pollution sources because of inexpensive land and property values, and lack of political power to influence traffic infrastructure and facility siting decisions (Ringquist, 2005). Consequently, residential segregation of low-income and racial/ethnic minority populations may also be linked to higher ambient pollution concentrations, such as ambient $PM_{2.5}$, compared to predominately affluent and white communities (Clark et al., 2014; Kravitz-Wirtz et al., 2016; Pastor et al., 2001). The extent to which the inequitable distribution of ambient pollution interacts with housing conditions that may further exacerbate exposure inequities needs to be further explored.

In this study we estimated AER as a measure of home leakiness for each residential parcel across Massachusetts using publicly-available data, and we used the results to understand how physical building characteristics of a residence can modify exposure to $PM_{2.5}$ of ambient origin. We further evaluated the role AER can play in exacerbating or ameliorating ambient $PM_{2.5}$ exposure inequalities. This work was conducted within the Center for Research on Environmental and Social Stressors in

Housing across the Life Course (CRESSH), a center that studies environmental health disparities in low-income communities and throughout Massachusetts.

Methods

The project was conducted in three phases: 1) calculate seasonal AER across all residential parcels (i.e. address level tax assessor parcels categorized as residential) in Massachusetts using an empirically-derived physical-based model parameterized with variables from public databases; 2) estimate concentrations of indoor $PM_{2.5}$ of ambient origin across all Massachusetts residential parcels by combining the calculated AERs and ambient $PM_{2.5}$ concentrations using an infiltration box model; and 3) perform an inequality analysis of residential parcel sociodemographic characteristics across different levels of AER and ambient $PM_{2.5}$ exposure groups.

Data Sources

Data sources used to parameterize the AER equation and perform the inequality analysis are listed in Table 3.1, and described in detail below. Housing, sociodemographic, and meteorological data used to parameterize the AER equations were obtained from public databases so as to provide a method that could be replicated in other communities in the US. Data were available at different geographical resolutions but all datasets were linked to the parcel using SAS software (version 9.3; SAS Institute Inc., Cary, NC) to summarize datasets, and ArcGIS to spatially join datasets (version 10.3; ESRI, Inc.).

Table 3.1. Data sources used in infiltration calculation and inequality analysis

Dataset	Variables	Coverage Year	Geographic Resolution	Equation Use	Publicly Available
Massachusetts Tax Assessor	Year built, number of stories, building area (m^2)	2009-2015	Parcel	Equations 3.1 and 3.2a-b, S3.1 & S3.2	X
Census	Racial and ethnic characteristics	2010	Block group	Equation 3.2a & 3.2b; Inequality analysis	X
American Community Survey	Socioeconomic characteristics	Average 5-year 2006-2010	Block group	Inequality analysis	X
Residential Energy Consumption Survey	Indoor temperature	2009	N/A	Equation 3.3	X
MassGIS Land Use	Categories of land use	2005	Point	Equation 3.3 and S3.1	X
Automated Surface Observing System	Average seasonal wind speed (m/s)	2010	Point	Equation 3.3	X
	Average seasonal temperature (◦C)	2010	Point	Equation 3.3	X
1 km^2 gridded surface $PM_{2.5}$ (Kloog et al., 2014)	Average seasonal ambient $PM_{2.5}$ ($\mu g/m^3$)	2010	1 km^2 grid	Equation 3.4; Inequality analysis	

Housing Characteristics. We obtained Level 3 Assessor's Parcel data from the Massachusetts Office of Geographic Information (MassGIS) and Metropolitan Area Planning Council (MAPC) (MassGIS, 2016; Metropolitan Area Planning Council, 2016). MassGIS standardizes parcel data from each town's assessor across the state and provides information on number of stories, square footage, property value, year built, property ownership, house style and number of rooms. MAPC compiled the individual town files into one file for the state. For this work, we restricted to residential parcels only. We assume each parcel contains one residential single- or multi-family building. Further details about data cleaning and missing data imputation procedures can be found in Section S3.1 and Table S3.1.

Demographic Data. We gathered information on race, ethnicity, income, educational attainment, and poverty status from the US Census and American Community Survey (ACS) at the block group (BG) unit of analysis. We obtained race and ethnicity data from Census 2010, and measures of income and educational attainment from ACS 2006-2010 5-year estimates. ACS and Census data were assigned to the parcel based on the BG where the parcel was located.

Meteorological and Land Use Data. Ambient surface temperature (°C) and wind speed (m/s) data were obtained from Automated Surface Observing System (ASOS) monitors located at airports, which are maintained by the National Weather Service, Federal Aviation Administration and Department of Defense. We averaged daily temperature and wind speed over winter (December 22nd 2009- March 21st 2010) and summer (June 21st 2010- September 22nd 2010) seasons, and assigned values to each parcel based on the nearest monitor.

We obtained land use classifications at 0.5 meter resolution from MassGIS (2005), who used semi-automated methods and digital ortho-imagery captured in April 2005 to classify land use into 40 separate categories. Parcels were assigned land use categories based on the land use polygon where the parcel was located.

Ambient $PM_{2.5}$. Surface $PM_{2.5}$ at a 1 km^2 resolution was obtained from an air pollution dataset that has been validated and used in previous studies of air pollution (Fleisch et al., 2016; Mehta et al., 2016; Shi et al., 2016). Details of the ambient $PM_{2.5}$ prediction models can be found in (Kloog et al., 2014). Briefly, this modeling approach used a combination of aerosol optical depth (AOD) satellite data retrieved using the multi-angle implementation of atmospheric correction (MAIAC) algorithm, land use, and meteorological predictors of variation in surface-$PM_{2.5}$ (i.e. percentages of high development and forest areas, elevation, population density), and outdoor monitor $PM_{2.5}$ concentrations to calculate a daily $PM_{2.5}$ estimate on a 1 km^2 grid (Kloog et al., 2014). This model produced an overall "out-of-sample" R^2 for daily values of 0.88, and cross validation results produced a slope of observed versus predicted of 0.99, demonstrating high predictive reliability of the model. For the purposes of the present study, we averaged gridded daily $PM_{2.5}$ concentrations ($\mu g/m^3$) over winter and summer seasons and assigned gridded values to each BG using the closest 1 km^2 grid centroid.

Air Exchange Rate Calculations.

We estimated AER using the Lawrence Berkeley National Laboratory (LBNL) physical-based infiltration model (Sherman and Grimsrud, 1980). This model was linked to an empirically-derived leakage area model estimated from measurements of 70,000 homes across the United States (Breen et al., 2010; Chan et al., 2005). The LBNL model predicts AER due to airflow through small unintentional openings in the building envelope such as holes and cracks. It does not take into account natural ventilation due to window and door openings, or mechanical ventilation. For the purposes of this study, we are interested only in leaks through unintentional cracks and openings in buildings as a measure of overall home leakiness. We employ a modified equation built by Sarnat et al. (2013) that estimates AER from the LBNL model exclusively for single family homes by applying it to estimate AER for both single family and multi-family homes (details found in the Supplement, Section S3.3) (Sarnat et al., 2013). To study seasonal differences, we calculated average AER for all residential parcels in Massachusetts for the winter and summer seasons. We chose these seasons to represent maximum potential variability in meteorological conditions. The AER equation was defined as:

$$AER = \frac{NL}{1000 \times H}\left[\frac{2.5}{H}\right]^{0.3} S \qquad \text{[Equation 3.1]}$$

where *NL* is the normalized leakage area of the building envelope (Chan et al., 2005); *H* is house height (calculated as number of stories times 2.5 meters plus 0.5 meters for the roof); and *S* represents the infiltration rate across the building envelope due to pressure differences, which are driven by indoor-outdoor temperature differences (stack effect) and wind (wind effect) (Sarnat et al., 2013).

NL was estimated using literature-reported regression parameters (Chan et al., 2005). This particular area leakage model is suitable for the purposes of this work because it can be parameterized using publically-available data sources. It has previously been found to perform equally well as alternative area leakage models (Mcwilliams and Jung, 2006). In this model, median year built and floor area (m^2) were the main *NL* predictors (Equation 3.2a & 3.2b). SF home building floor area values were taken directly from the assessor's database parcel data, and we calculated floor area of a representative unit in a multi-family home by dividing building area by the number of units per floor. Chan et al. (2005) predicts NL separately for residents earning 125% below the federal poverty line (NL_a) and all other homes (NL_b). Regardless of year built and floor area, homes below the poverty line from the database used in Chan et al (2005) were leakier than conventional homes, indicating residual neighborhood and individual-level characteristics above and beyond the age and size of the home that influence home leakiness. We predicted NL_a for parcels that fell within a BG categorized as a census-defined poverty area, where at least 20 percent of the households are below the poverty threshold (Bureau of the US Census, 1995). *NL* parameters for low-income and conventional homes are shown below:

$$NL_a = e^{11.1 + (-0.00537\ x\ median\ year\ built)\ +\ (-0.00418\ x\ m^2)} \quad \text{[Equation 3.2a]}$$

$$NL_b = e^{20.7 + (-0.0107\ x\ median\ year\ built)\ +\ (-0.0022\ x\ m^2)} \quad \text{[Equation 3.2b]}$$

The infiltration parameter, *S* is defined as:

$$S = \sqrt{f_s^2\ x\ |T_{in} - T_{out}| +\ f_w^2\ x\ u^2} \quad \text{[Equation 3.3]}$$

where T_{in} is the home indoor temperature, assumed to be 20°C in the winter months and 22°C in the summer months (U.S. Energy Information Administration, 2016); T_{out} is the seasonal mean ambient temperature at the closest monitor to the parcel; *u* is the seasonal mean wind speed (m/s) averaged from daily observed wind speeds between 2009 and 2010 at the closest monitor to the parcel; f_s is the stack coefficient; and f_w is the wind coefficient. Details for f_s and f_w estimation can be found in the Supplement (S3.2 and Table S3.2).

Indoor $PM_{2.5}$ of Ambient Origin Concentration Calculations.

To estimate concentrations of indoor $PM_{2.5}$ of ambient origin across all Massachusetts residential parcels, we combined the calculated seasonal AERs and ambient $PM_{2.5}$ concentrations using a standard single-compartment infiltration box model, as has been done in previous studies (Baxter et al., 2007; Fabian et al., 2012a; Long et al., 2001). Because our goal was to determine how seasonal AER – based on housing characteristics – modified indoor $PM_{2.5}$ concentrations of ambient sourced $PM_{2.5}$, we ignored indoor $PM_{2.5}$ sources. The equation for indoor $PM_{2.5}$ concentrations of ambient origin (C_{in} in μg/m^3) is:

$$C_{in} = \frac{Pa}{a+k} C_{Out} \qquad \text{(Equation 3.4)}$$

where P=penetration efficiency (dimensionless), a=AER (h^{-1}) assigned at a parcel level in the winter and the summer, calculated from Eq. 1, k= $PM_{2.5}$ decay rate (h^{-1}), and C_{out} = outdoor ambient $PM_{2.5}$ concentration (μg/m^3). Based on literature-reported parameters estimated previously in Breen et al. (2014) from Environmental Protection Agency Panel study data, we assumed that P=0.84 and k=0.21 h^{-1} (Breen et al., 2015). The P and k values estimated in Breen et al. (2015) are consistent with previously reported estimates over a variety of housing stock and geographic regions (Burke et al., 2001; Meng et al., 2005; Thatcher et al., 2003). As a sensitivity analysis, we test the lower and upper confidence limits of P and k estimated in Breen et al. (2015) (Breen et al., 2015). These values are reported in Table S3.5, found in Supplement. C_{out} was assigned to each residential parcel from the corresponding 1 km^2 $PM_{2.5}$ data described above.

AER and Ambient $PM_{2.5}$ Inequality Analysis

We conducted an inequality analysis of our estimated AER and ambient $PM_{2.5}$ concentrations to understand whether AER modifies the extent to which different population groups are exposed to $PM_{2.5}$ of ambient origin. We defined "high-exposure" parcels as those with both AER and ambient $PM_{2.5}$ above the 90th percentile, and "low-exposure" parcels as those with both AER and ambient $PM_{2.5}$ below the 10th percentile. We then assigned parcels demographic characteristics based on the BGs in which they fell, and we calculated summary statistics for the demographic characteristics for the combined ambient $PM_{2.5}$ and AER upper and lower deciles. We also examined the demographics for each of the upper and lower deciles of AER and ambient $PM_{2.5}$ exposure independently, to determine the drivers of the combined patterns. In addition, we examined the lower decile of ambient $PM_{2.5}$ and the upper decile of AER (low-$PM_{2.5}$ areas with high infiltration), and the upper decile of ambient $PM_{2.5}$ and the lower decile of AER (high--$PM_{2.5}$ areas with low infiltration). We further stratified by urban or rural BG classification, based on Census classifications (Ratcliffe et al., 2016).

Results

Air Exchange Rates

We estimated AER for 1,659,098 residential parcels (77% of total parcels) in Massachusetts and calculated summary statistics stratified by housing type (Table 3.2). SF homes were built most recently (median: 1960), while small apartment buildings have the oldest median year built (1900). Median floor areas for a representative unit within multi-family buildings are smallest for units in both small (71.8 m^2) and large (80.7 m^2) multi-family buildings, while SF homes have the largest median building area (212.7 m^2). Of 22,387 small apartment buildings, 40.1% are categorized as low-income, whereas only 6.5% of SF households are categorized as low-income (Table S3.3). Among multi-family buildings, large apartment building parcels have the highest percentage of conventional (i.e., not low-income) parcels (82.9%), followed by duplex/triplex parcels (70.6%) and small apartment building parcels (59.9%) (Table S3.3).

Density of surrounding obstructions increased by increasing shelter class (Table

S3.2). The largest percentage of low-density parcels is among SF homes (15%), and the majority of duplex/triplex parcels are located in very high-density areas. NL rates were highest in small apartment buildings and lowest in SF homes.

Table 3.2. Residential housing characteristics and seasonal air exchange rates, stratified by housing type

	Year Built			Height (m)			Building Area (m^2)			NL		
	25th	50th	75th	25th	50th	75th	25th	50th	75th	25th	50th	75th
Total (n=1,659,098)	1920	1955	1978	3.0	3.0	5.5	131.2	194.5	280.0	0.37	0.54	0.79
Single Family (n=1,383,249)	1935	1960	1983	3.0	4.6	5.5	143.3	212.7	300.3	0.35	0.49	0.66
Duplex/Triplex (n=207,722)	1900	1907	1923	3.0	3.0	3.0	109.1	146.2	191.3	0.80	0.97	1.15
4-8 Apartment Buildings (n=22,387)	1900	1900	1920	3.0	3.0	3.0	71.8	90.0	113.5	1.03	1.21	1.51
>8 Apartment Buildings (n=45,740)	1900	1945	1987	3.0	3.0	3.0	80.7	107.4	142.6	0.45	0.84	1.12

*All duplex/triplex and apartment building units are assumed to have a height of one story (3 meters); building area reflects the individual unit for multi-family homes.

Table 3.3 Residential air exchange rates, stratified by housing type

	AER (h^{-1}), winter			AER (h^{-1}), summer		
	25th	50th	75th	25th	50th	75th
Total (n=1,659,098)	0.47	0.74	1.09	0.23	0.37	0.57
Single Family (n=1,383,249)	0.42	0.67	0.94	0.22	0.34	0.51
Duplex/Triplex (n=207,722)	1.09	1.39	1.78	0.45	0.67	0.97
4-8 Apartment Buildings (n=22,387)	1.21	1.42	1.76	0.43	0.54	0.68
>8 Apartment Buildings (n=45,740)	0.54	1.00	1.34	0.22	0.37	0.57

Table 3.3 displays summary statistics for AER across Massachusetts. SF homes have the lowest median AER for both winter (0.67 h^{-1}) and summer (0.34 h^{-1}) months, while units in small apartment buildings have the highest median AER in the winter (1.42 h^{-1}), and duplex/triplex homes have the highest median AER in the summer (0.67 h^{-1}). AER distributions from imputed data were similar to values computed using complete case parcels for all housing types (Table S5).

Indoor $PM_{2.5}$ Concentrations

Table 3.4 compares outdoor ambient $PM_{2.5}$ concentrations and indoor $PM_{2.5}$ concentrations originating from ambient $PM_{2.5}$. Using the single-compartment box model, we found that indoor concentrations ranged from 0.40 to 9.0 $\mu g/m^3$, and were on average 3 $\mu g/m^3$ lower than outdoor concentrations. Highest mean ambient outdoor $PM_{2.5}$ concentrations were among apartment buildings with 4-8 units in both winter (9.8 $\mu g/m^3$) and summer (7.9 $\mu g/m^3$) months. The highest mean indoor concentrations were among apartment buildings with 4-8 units in the winter (7.1 $\mu g/m^3$) and among duplex/triplex homes in the summer (4.9 $\mu g/m^3$). High indoor $PM_{2.5}$ may be reflective of the higher AERs estimated for these two housing types, as compared to other housing types. We additionally found that the coefficient of variation for indoor $PM_{2.5}$ concentration across all parcels is more than twice that of ambient outdoor parcel-level $PM_{2.5}$ concentration, with greater variability in the winter than summer, demonstrating that housing characteristics increase the variability in ambient $PM_{2.5}$ exposure across the population.

Table 3.4. Indoor $PM_{2.5}$ concentration of ambient origin compared to outdoor ambient $PM_{2.5}$ concentration across all Massachusetts parcels

		Winter Ambient $PM_{2.5}$ ($\mu g/m^3$)					Summer Ambient $PM_{2.5}$ ($\mu g/m^3$)				
		mean	SD	25th	75th	CV	mean	SD	25th	75th	CV
Total (n=1,659,098)	Outdoor	9.2	0.8	8.8	9.8	0.09	7.3	0.8	6.8	7.8	0.11
	Indoor	5.9	1.1	5.2	6.7	0.19	3.8	1.0	3.1	4.6	0.27
Single Family (n=1,383,249)	Outdoor	9.2	0.8	8.7	9.7	0.09	7.2	0.8	6.7	7.7	0.11
	Indoor	5.7	1.1	5.0	6.4	0.19	3.6	1.0	3.0	4.4	0.27
Duplex/Triplex (n=207,722)	Outdoor	9.7	0.6	9.5	10.0	0.06	7.8	0.6	7.6	8.1	0.07
	Indoor	7.0	0.6	6.7	7.5	0.09	4.9	0.8	4.4	5.5	0.17
4-8 Apartment Buildings (n=22,387)	Outdoor	9.8	0.6	9.5	10.2	0.07	7.9	0.6	7.6	8.2	0.07
	Indoor	7.1	0.6	6.8	7.5	0.09	4.7	0.7	4.3	5.2	0.15
>8 Apartment Buildings (n=45,740)	Outdoor	9.6	0.9	9.2	10.1	0.09	7.5	0.9	7.2	8.1	0.11
	Indoor	6.4	0.9	5.7	7.2	0.15	3.9	1.0	3.1	4.8	0.26

$PM_{2.5}$ and air exchange rate exposure inequality analysis

Parcels in the highest ambient $PM_{2.5}$ and AER quantiles are located in BGs with higher percentages of non-white populations, low-income and low educational-attainment populations. Figure 3.1 displays estimated winter AERs at the parcel level, average winter ambient $PM_{2.5}$ assigned to each residential parcel, and Census demographic characteristics at BG resolution in an area of Eastern Massachusetts. The highlighted urban area of Figure 3.1 demonstrates that BGs containing parcels with the highest AER quantile also contain parcels with the highest $PM_{2.5}$ values. These same BGs also tend to have greater percentages of Hispanic and low-income populations, as compared to parcels with low AER and low $PM_{2.5}$.

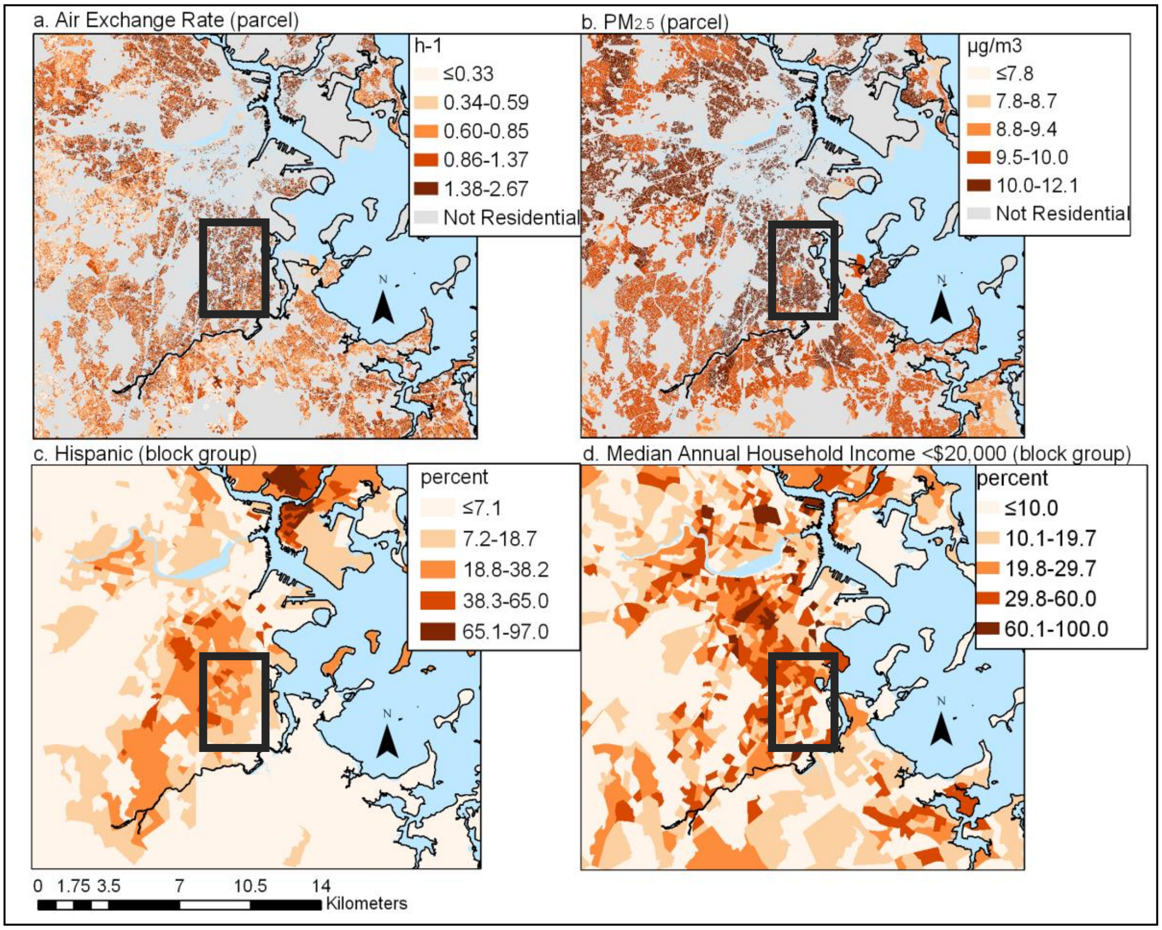

Figure 3.1. Map of Eastern Massachusetts in 2010 showing distribution of a) winter air exchange rates at parcel level, b) winter $PM_{2.5}$ concentrations at parcel level, c) % Hispanic at block-group, and d) % median annual household income below $20,000 at block group.

Figure 3.2 shows the sociodemographic characteristics (i.e. racial/ethnic, median household income, and educational attainment categories) of the block groups containing the high-exposure (>=90th percentile of AER or $PM_{2.5}$ distributions, separately) residential parcels compared to low-exposure parcels (<=10th percentile of AER or $PM_{2.5}$ distributions, separately). We present results averaged across winter and summer seasons, as results did not vary seasonally (seasonal results not shown).

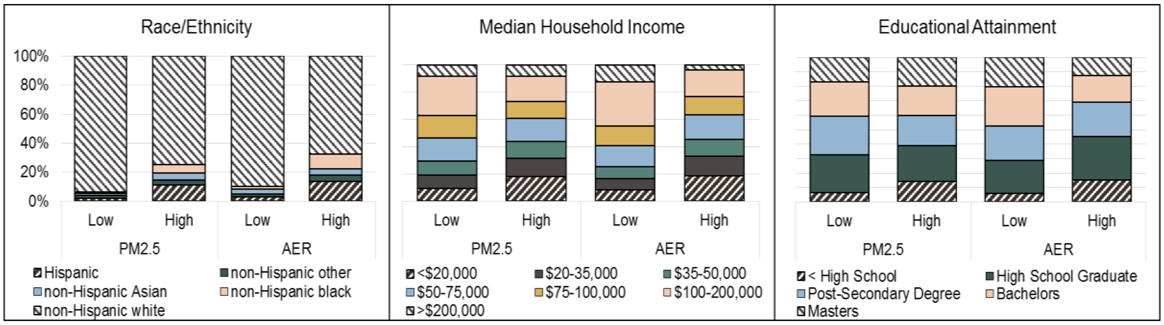

Figure 3.2. Sociodemographic characteristics of block groups containing the residential parcels with the lowest (<=10th %tile) and highest (>=90th %tile) air exchange rates (AER) and ambient $PM_{2.5}$. Source: US Census 2010

For this analysis, BGs containing parcels with high ambient $PM_{2.5}$ have a lower percentage non-Hispanic white population (75%) than low ambient-$PM_{2.5}$ exposed parcels (94%). They also contain smaller proportions of homes with median household incomes greater than $75,000 per year (39% vs. 53%). Patterns for educational attainment are more complex, with BGs containing high $PM_{2.5}$–exposed parcels having a greater percentage of both residents without a high school degree and residents with a graduate degree.

Similarly, BGs containing parcels with high AER also have a lower percentage non-Hispanic white population (68%), versus 90% in BGs containing low AER parcels. Among the non-white populations, BGs containing high-$PM_{2.5}$ parcels were 6% non-Hispanic black, 5% non-Hispanic Asian, 3% non-Hispanic other, and 11% Hispanic. BGs containing high-AER parcels were 10%, 5%, 4% and 14% non-Hispanic black, non-Hispanic Asian, non-Hispanic other and Hispanic, respectively. These demographic trends were generally similar for income and educational attainment groups, with BGs containing higher-AERs parcels having higher average proportions of low-income households and low-educational attainment populations compared to BGs containing low-AER parcels.

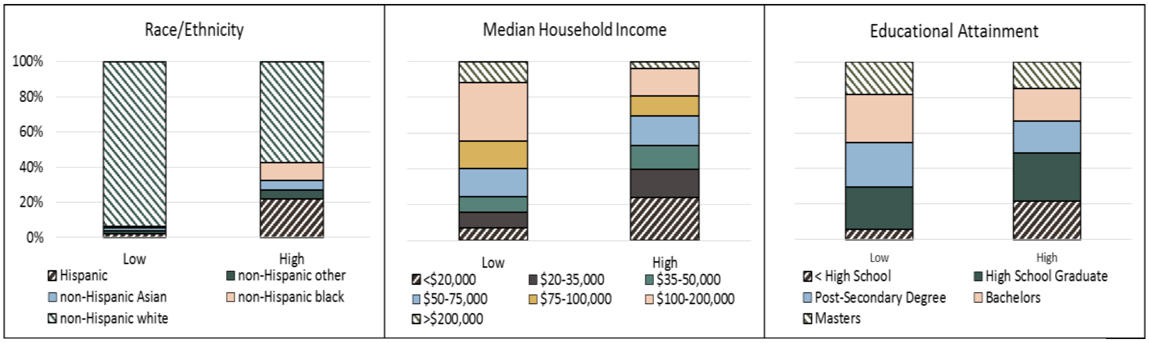

Figure 3.3. Sociodemographic characteristics of block groups containing the residential parcels with the lowest air exchange rates (AER) in areas with the lowest ambient $PM_{2.5}$ (low-exposure, <10th %tile) versus block groups containing parcels with the highest AER and $PM_{2.5}$ (high-exposure, >90th %tile)**.** Source: US Census 2010.

Figure 3.3 shows the sociodemographic characteristics (i.e. racial/ethnic, median household income, and educational attainment categories) of the block groups containing the high-exposure (>=90th percentile of *both* AER and $PM_{2.5}$) residential parcels compared to the low-exposure parcels (<=10th percentile of *both* AER and $PM_{2.5}$).

High-exposure parcels are located in BGs with a higher percentage Hispanic and non-white individuals, low-income households, and individuals with lower educational attainment than BGs containing low-exposure parcels. Specifically, whereas low-exposure parcels are located in BGs that are, on average, 6.4% non-white, high-exposure parcels are located in BGs that are 42% non-white (22% Hispanic, 6% non-Hispanic Asian, 10% non-Hispanic black, 5% other non-Hispanic races). Low-exposure parcels are located in BGs with, on average, 7% of households with a median annual income below $20,000, versus 24% for high-exposure parcels. Similarly, the low-exposure parcels are located in BGs with 6% of individuals with less than a high school education, versus 22% in BGs containing the high-exposure parcels.

Parcels characterized as being in both the low $PM_{2.5}$ and high AER distributions are located in BGs with similar racial and income characteristics as parcels characterized as high $PM_{2.5}$ and low AER (Figure S3.1). When stratified by urban and rural BGs, we find that the 90th percentile ambient $PM_{2.5}$ and AER parcels that are located in BGs with higher percentage non-white populations are mostly characterized as urban BGs, rather than rural, reflecting more racial/ethnic inequality in home "leakiness" and ambient $PM_{2.5}$ exposure among urban compared to rural geographies (Figure S3.2). Patterns are similar among income and educational attainment population groups, where the high exposure parcels located in urban BGs contain higher proportions of vulnerable populations (low-income and low educational attainment) compared to low-exposure (10th percentile of AER and ambient $PM_{2.5}$) and rural BGs.

Discussion

Estimating AER across Massachusetts using Census demographic data at the BG level and housing characteristics at the parcel level allows for a straightforward estimation of AER over large geographic regions, housing types, and populations to better characterize the relationship between housing characteristics and outdoor ambient air pollution exposure. These methods and public data can be extrapolated to any area in the US, and can be extended to estimate indoor concentrations of a variety of outdoor-generated air pollutants (e.g., NO_2, CO). Additionally, these data sources provide a unique opportunity to examine inequalities at a fine spatial resolution, and to characterize exposure inequalities that are not solely based on ambient concentrations.

To our knowledge, no previous studies have examined exposure inequality as a consequence of combined AER and ambient air pollution exposure. Low SES populations tend to live in homes of lower value, which may be reflective of proximity to pollution sources, of the quality of the home itself, or other neighborhood factors. Residential segregation, and resultant housing conditions, are closely linked to community environmental health (Baxter et al., 2007; Rauh et al., 2008). Using spatially and temporally resolved estimates of $PM_{2.5}$ concentrations and AER to analyze exposure inequality, we found that neighborhoods that contain parcels with both high ambient $PM_{2.5}$ and AER disproportionately include Hispanic or non-white, low income and low educational attainment populations.

The overall demographic makeup of Massachusetts in 2010, according to U.S. Census 2010 data, was 74% non-Hispanic white, 7% non-Hispanic black, 6% non-Hispanic Asian, 3% non-Hispanic other and 10% Hispanic. When we compare these demographic distributions to those of BGs containing high-exposure parcels when $PM_{2.5}$ and AER are overlaid, we see that non-white populations, in particular Hispanic populations, are disproportionately burdened with leakier homes located in locations with higher ambient $PM_{2.5}$ concentrations. Stratified analysis also confirms our *a priori* hypothesis that marginalized populations experience a cumulative burden of both high AER and high ambient air pollution concentrations, and that these inequalities are magnified when AER and ambient $PM_{2.5}$ are overlaid. The wealth of existing studies examining exposure inequality to pollutants of ambient origin do not incorporate measures of the home microenvironment that modify indoor exposures (Bell and Ebisu, 2012; Clark et al., 2014; Morello-Frosch and Jesdale, 2006). Our findings demonstrate that exposure inequalities found in these previous studies may be compounded when housing characteristics are considered.

Variations in AER between housing types estimated in our study are attributed to differences in unit volume, age of the home, weatherization, income parameters and the surrounding terrain. Higher AERs estimated for units in large apartment buildings are likely explained by smaller average floor areas and a greater percentage of parcels categorized as "low-income" (40%) within this housing type. Duplex and triplex parcels had relatively high AERs compared to SF homes and units in large apartment buildings, which also may be due, in part, to a large percentage categorized as low-income and older construction, both of which are expected to increase the AER estimates.

The AER results found in this study are in agreement with published AER values, estimated through various modeling techniques and field measurements for use in exposure characterization or epidemiological analysis (Baxter et al., 2007; Breen et al., 2010; Meng et al., 2005; Persily et al., 2010; Sarnat et al., 2013; Zota et al., 2005). Using a modified version of the LBNL model, Sarnat et al. 2013 estimated AERs at the zip code level in Atlanta, Georgia. The overall AER mean (min-max) was 0.27 h^{-1} (0.03-1.04) for all study-area zip codes. Higher average SF AERs estimated in our study as compared to Sarnat et al. (2013) may be attributed to larger homes and smaller indoor-outdoor temperature differences in Atlanta compared to Massachusetts (Sarnat et al., 2013). Persily et al. (2010) used the CONTAM multizone network airflow model to estimate AER across 209 SF and multi-family dwellings in the United States. Median AER for New England detached homes was 0.44 h^{-1} (10^{th}-90^{th}: 0.22-1.18) (Persily et al., 2010).

Median AER for apartments built before 1940 was 0.31 h^{-1} (10^{th}-90^{th}: 0.16-0.72) and for apartments built after 1990 the median AER was 0.14 h^{-1} (10^{th}-90^{th}: 0.07-0.31). Our estimates were similar compared to homes modeled as SF and detached, but apartment estimates were generally higher in our study than simulated using the CONTAM model. These differences are likely because of the ability of CONTAM to include information about corridors and ventilation systems, which weighted newer apartment buildings with ventilation systems towards the lower end of the AER distribution (Persily et al., 2010).

As for studies that measured AER, Yamamoto et al. 2009 used tracer gas techniques to measure AER in 500 homes in three US metropolitan areas. Median measured AERs in Los Angeles County, Elizabeth, New Jersey, and Houston, Texas were 0.87, 0.88, and 0.47 h^{-1}, respectively (Yamamoto et al., 2010). Zota et al. (2005) measured AERs in urban Boston public housing apartments (n=77) using the perfluorocarbon tracer (PFT) method. During the heating season median AER was 0.49 h^{-1} (range: 0.08-1.40) and 0.85 h^{-1} (0.14-2.23) during the non-heating season (Zota et al., 2005). These findings are inconsistent with higher estimated AERs in the heating versus the non-heating season found in our study, which is presumably because of window-opening behavior, HVAC system and window AC use that increase AER in the summer months.

Although we were unable to validate our modeled AER estimates against measured AER across the state, previous studies that have employed the LBNL model have assessed its validity (Baxter et al., 2016; Breen et al., 2010). Breen et al. 2010 compared LBNL estimates to daily 24 hour AER measurements using the PFT method in

31 North Carolina detached homes. The median absolute difference between LBNL-estimated AERs and measured AERs was 40% (0.17 h^{-1}), with estimated AERs slightly underpredicting measured values, also likely due to window-opening behavior. The mean modeled AERs (10^{th}, 90^{th}) were similar to our results: 0.26 h^{-1} (0.14, 0.40) and 0.62 h^{-1} (0.37, 0.86) in the summer and winter, respectively. Baxter et al. (2016) stochastically estimated residential AERs using the LBNL model across four US cities, and incorporated Census-tract level information on AC prevalence and window activity. The authors compared results using publicly versus study-specific collected data. Comparing LBNL-modeled to measured AER in Elizabeth, NJ, respectively, AER was 1.03 h^{-1} (range: 0.8-1.28) and 1.60 h^{-1} (range: 0.33-4.47) during the coldest days, and 0.94 h^{-1} (0.62-1.13) versus 0.89 h^{-1} (0.11-1.33) on the warmest days (Baxter et al., 2016). We can conclude from these studies that estimated AER using the LBNL model is a close approximation of measured AER across various study areas, at different geographic resolutions and over different temporalities.

Using estimated AERs and $PM_{2.5}$-specific assumptions about penetration efficiency and decay rate, we calculated indoor $PM_{2.5}$ of ambient origin across all Massachusetts parcels, demonstrating that $PM_{2.5}$ exposure variability increases by a factor of two when housing characteristics are considered. The contribution of outdoor ambient pollutants to indoor concentrations has been mixed (Baxter et al., 2013a, 2013b, 2007b; Chen et al., 2012; Clougherty et al., 2008; Ozkaynak et al., 2013; Zota et al., 2005). However, evidence suggests that effect modification of ambient-pollution related health outcomes are associated with daily (Sarnat et al., 2013) and overall (Bell and

Dominici, 2008; Chen et al., 2012, 2011; Levy et al., 2005) changes in AER. As an example, Shi et al. 2017 evaluated bias in health effect estimates from using ambient concentration versus personal residential probability-distributed annual and seasonal particle infiltration in Beijing. By comparing $PM_{2.5}$ infiltration factors to ambient exposures, the authors found that on average, residences contained 56% of ambient $PM_{2.5}$ in the indoor environment (Shi et al., 2017), consistent with our estimates.

Air pollution epidemiological studies that apply AER as a covariate or modifying factor have produced less exposure measurement error, and thus, more precise effect estimates of associations between residential exposure to ambient air pollution and health outcomes, compared to traditional analyses (Baxter et al., 2013b; Dionisio et al., 2016). The application of AERs to estimate outdoor-generated indoor pollutant concentrations can be incorporated into future epidemiological studies of ambient air pollution exposure to refine effect estimates. Previously published health studies have found that effect estimates of O_3 and NO_2 with various health outcomes are even more sensitive than ambient $PM_{2.5}$ to the modifying effects of AER, highlighting the importance of co-pollutant approaches in air pollution analyses (Baxter et al., 2007; Sarnat et al., 2013).

Our study had some limitations related to simplifying assumptions used to calculate AER. The LBNL model used for this study only takes into account infiltration due to cracks and openings in the building envelope. It does not consider occupant home operation or activities such as window opening, air conditioner status, air filter use, or cooking, nor does it consider variability in home characteristics from mechanical ventilation. Consequently, our approach does not capture variability and extreme values due to unmeasured occupant behavior and mechanical ventilation, leading to potential underestimation in our estimates, consistent with previous studies (Breen et al., 2015; Logue et al., 2015). However, previous studies in central North Carolina (Breen et al., 2010) and Detroit, Michigan (Breen et al., 2014) have demonstrated no substantial difference in measured and modeled (using the LBNL model and the extended LBNL model that incorporates window-opening behavior) AER for days with open windows compared to days with closed windows. We therefore believe the LBNL model is appropriate for our analysis, assuming closed windows (Breen et al., 2010). Validated models to estimate AER in multi-family homes and publicly available information on occupant behaviors and mechanical ventilation are needed to further refine AER estimates and account for variability in exposure across study populations.

We modeled condominiums and apartments located in larger buildings as individual, unattached homes, assuming that each unit was a single, well-mixed compartment, so we were neither able to account for their location and elevation within a given building nor for the complex multi-zone characteristics of these buildings. Also, multi-family units were all assigned shelter class “5” to account for walls that were not outside facing, which may overestimate AER for some these units. Another limitation is in the NL estimation, which was derived from a nationwide survey, and may not be representative of the Massachusetts housing stock. Categorization of homes as low-income or conventional were based on BG level, rather than parcel-level characteristics. Additionally, because we were unable to examine the surrounding terrain of each home, we used land use characterization to assign terrain class. Due to lack of applicable equations that predict indoor temperature, we used uniform indoor temperature values based on RECS data, which can reduce variability in the stack effect and the resulting AER. The ambient $PM_{2.5}$ concentrations used in the present analysis were predicted using a novel air quality model developed by Kloog et al. 2014 (Kloog et al., 2014). Because the model requires many variables, its application may be limited to regions where the necessary public data is available.

Though beyond the scope of this study, our focus on air pollution of ambient origin omits the potential influence of indoor sources on personal exposure. While homes with low AER will have reduced infiltration of ambient outdoor-generated $PM_{2.5}$, they will have an enhanced influence from any indoor sources (e.g. combustion). As such, our study only characterizes the most highly exposed subpopulations to ambient air pollution across Massachusetts. That said, a focus on air pollution of ambient origin is consistent with interpretation of epidemiological evidence based on central site monitors and provides the opportunity to test effect modifiers in future epidemiological analyses. Our analytical framework would allow for separate examination of the influence of indoor sources on patterns of personal exposure given the requisite source information. Our modeling approach can be expanded to any area in the US, and can be used in epidemiological studies to refine air pollution exposure modifiers or to characterize exposure disparities that are not solely based on ambient concentrations.

Supplement

S3.1: Parcel imputation procedure

In order to estimate air exchange rate (AER) for all residential parcels in Massachusetts, we created a complete dataset of Massachusetts Assessor's database variables by imputing missing parcel data by the Markov Chain Monte Carlo multivariate normal model (Schafer, 1997). We assessed the missing at random assumption by examining missing data patterns. Data were log-transformed within the imputation procedure to fulfill the normality assumption. Missing parcel data were 11%, 19%, and 17% for building area, number of stories and year built, respectively. Using PROC MI, we created 15 imputed datasets as recommended by Graham et al (Graham et al., 2007). Details on variables included as predictors of missing observations and percent missingness can be found in Table S3.1.

Table S3.1. Variables imputed and included in multiple imputation procedure for all Massachusetts parcels

Variable	Percent Missing
Lot Area	0
Percent Impervious	0.07
Square Meter Paved	0.08
Percent Paved	0.08
Percent Building	0.16
Total Value per Acre	0.32
Building Value	1.2
Square Meters Impervious	1.8
Number of Estimated Units*	1.9
Land Value	2.0
Building/Land Ratio	2.2
Floor/Area Ratio	2.5
Square Meter Building	4.0
Year Built*	6.7
Building Value per Square Foot	7.8
Number of Stories*	8.6
Building Area*	10.2
Number of Rooms	16.8

*Variables used in AER equations

S3.2: Stack Coefficient and Wind Coefficient Estimation

Parameters calculated for the infiltration parameter S (Eq. 3) included:

f_s is the stack coefficient, estimated as:

$$\sqrt{\frac{1+\frac{Rfac}{2}}{3} x\ 1 - \left(\frac{x_{fac}^2}{2-R_{fac}^2}\right)^{\frac{3}{2}} x\ grav\ x\ \frac{H}{T_{ref}}} \qquad \text{[Equation S3.1]}$$

where R_{fac} is the fraction of total leakage from the floors and ceilings (assumed to be 0.5 for homes built before 2011 and 0.25 for homes built on or after 2011 (Ashrae, 2009; Sarnat et al., 2013; US Department of Energy, 2011)); X_{fac} is the difference between the leakage from a ceiling compared to that from a floor (assumed to be 0.25 (Sarnat et al., 2013)); *grav* is the earth's gravitational force (9.8 m/s^2) ; and T_{ref} is 298 K from the ideal gas law;

f_w is the wind coefficient, estimated as:

$$C_{fac}\ x\ (1 - R_{fac})^{\frac{1}{3}}\ x\ A_{fac}\ x\ \left(\frac{H}{10}\right)^{B_{fac}} \qquad \text{[Equation S3.2]}$$

where C_{fac} is set to reported values developed by LBNL based on local wind shielding from surrounding obstructions (shelter class) and house height (Sarnat et al., 2013). A_{fac} *and* B_{fac} are also factors developed by LBNL related to the geophysical terrain around the residence, and chosen based on shelter class.

Shelter class is a surrogate of wind shielding from surrounding obstructions, which we determined based on the land use classification in which each parcel was located. Shelter class was assigned based on the following: very low density residential (class 2), low density residential (class 3), medium density residential (class 4) and multi-family and high density residential (class 5).

S3.3 Applying LBNL Model to Multi-Family Units

To apply the LBNL model to a given residential unit in a multi-family building, we assigned all parcels categorized as multi-family a shelter class of “5.” This approach decreases the influence of the wind effect by maximizing the density of surrounding obstructions to account for apartment units having fewer externally-facing walls as compared to single family homes and duplex/triplex units. Shelter class definitions based on Sherman et al. (1980) can be found in Table S3.2.

Table S3.2. Shelter class B_{fac} and A_{fac} Parameters used in AER calculation (Sherman and Grimsrud, 1980)

Shelter Class	B_{fac}	A_{fac}	Description
1	0.10	1.30	No obstructions or local sheltering
2	0.15	1.00	Flat terrain with some isolated obstacles
3	0.20	0.85	Rural areas with low buildings, trees, etc.
4	0.25	0.67	Urban, industrial or forest area
5	0.35	0.47	Center of large city

For C_{fac} inputs see Tables 2.1-2.4 in Sherman et al. 1980 (Sherman and Grimsrud, 1980)

Table S3.3. Residential parcel frequencies for Equations 2a, 2b, S1 and S2

	Total (n=1,659,098)		Single Family (n=1,383,249)		Duplex/Triplex (n=207,722)		4-8 Apartment Buildings (n=22,387)		>8 Apartment Buildings (n=45,740)	
NL Category (n (%))										
Low Income	168,346	10.2	90,458	6.5	61,093	29.4	8,970	40.1	7,825	17.1
Conventional	1490,752	89.9	1,292,791	93.5	146,629	70.6	13,417	59.9	37,915	82.9
Shelter Class (n (%))										
1	210,236	12.7	205,813	14.9	4,423	2.1				
2	283,991	17.1	275,642	19.9	8,349	4.0				
3	546,564	32.9	509,177	36.8	37,387	18.0				
4	477,791	28.8	392,617	28.4	85,174	41.0				
5	140,516	8.5			72,389	34.9	22,387	100	45,740	100

Table S3.4. AER (h^{-1}) estimates complete case data

	AER (h^{-1}), winter					AER (h^{-1}), summer				
	mean	SD	25th	50th	75th	mean	SD	25th	50th	75th
Total	0.85	0.52	0.47	0.74	1.10	0.45	0.31	0.23	0.37	0.57
Single Family	0.74	0.44	0.42	0.67	0.94	0.40	0.27	0.21	0.34	0.50
Duplex/Triplex	1.47	0.55	1.09	1.39	1.78	0.76	0.41	0.45	0.67	0.97
4-8 Apartment Buildings	1.46	0.41	1.22	1.42	1.74	0.57	0.20	0.43	0.54	0.68
>8 Apartment Buildings	0.92	0.48	0.50	0.87	1.29	0.38	0.22	0.20	0.33	0.54

Table S3.5 Sensitivity analysis estimating indoor $PM_{2.5}$ of ambient origin for different values of penetration efficiency (*P*) and decay rate (*k*) as reported in Breen et al. (2015)[a]

		Winter Ambient $PM_{2.5}$ (µg/m^3)					Summer Ambient $PM_{2.5}$ (µg/m^3)				
		mean	SD	25th	75th	CV	mean	SD	25th	75th	CV
Total (n=1,659,098)	Indoor (lower CI)	5.7	0.9	5.1	6.3	0.16	3.9	0.9	3.3	4.5	0.22
	Indoor (upper CI)	6.0	1.3	5.1	7.0	0.22	3.7	1.2	2.9	4.6	0.31
	Indoor (lower CI *P*, upper CI *k*)	4.8	1.1	4.1	5.5	0.22	3.0	0.9	2.3	3.6	0.31
	Indoor (upper CI *P*, lower CI *k*)	7.1	1.1	6.4	7.9	0.16	4.9	1.1	4.2	5.7	0.22
Single Family (n=1,383,249)	Indoor (lower CI)	5.5	0.9	5.0	6.1	0.16	3.7	0.8	3.2	4.3	0.22
	Indoor (upper CI)	5.7	1.3	4.9	6.7	0.22	3.5	1.1	2.8	4.3	0.30
	Indoor (lower CI *P*, upper CI *k*)	4.6	1.0	3.9	5.3	0.22	2.8	0.9	2.2	3.4	0.30
	Indoor (upper CI *P*, lower CI *k*)	6.9	1.1	6.2	7.7	0.16	4.7	1.0	4.0	5.5	0.22
Duplex/Triplex (n=207,722)	Indoor (lower CI)	6.5	0.5	6.3	6.9	0.08	4.7	0.6	4.4	5.2	0.13
	Indoor (upper CI)	7.4	0.8	7.0	7.9	0.10	4.9	1.0	4.3	5.7	0.19
	Indoor (lower CI *P*, upper CI *k*)	5.9	0.6	5.6	6.3	0.10	3.9	0.8	3.4	4.5	0.19
	Indoor (upper CI *P*, lower CI *k*)	8.2	0.6	7.9	8.6	0.08	6.0	0.8	5.5	6.5	0.13
4-8 Apartment Buildings (n=22,387)	Indoor (lower CI)	6.6	0.5	6.4	6.9	0.08	4.7	0.6	4.3	5.0	0.12
	Indoor (upper CI)	7.5	0.7	7.2	8.0	0.10	4.7	0.8	4.3	5.2	0.17
	Indoor (lower CI *P*, upper CI *k*)	6.0	0.6	5.7	6.4	0.10	3.8	0.6	3.4	4.2	0.17
	Indoor (upper CI *P*, lower CI *k*)	8.3	0.7	8.0	8.7	0.08	5.8	0.7	5.5	6.3	0.12
>8 Apartment Buildings (n=45,740)	Indoor (lower CI)	6.1	0.7	5.6	6.7	0.12	4.0	0.9	3.4	4.7	0.21
	Indoor (upper CI)	6.6	1.1	5.7	7.6	0.17	3.9	1.1	2.9	4.8	0.29
	Indoor (lower CI *P*, upper CI *k*)	5.3	0.9	4.6	6.0	0.17	3.1	0.9	2.3	3.8	0.29
	Indoor (upper CI *P*, lower CI *k*)	7.7	0.9	7.0	8.4	0.12	5.1	1.1	4.2	5.9	0.21

[a]Jackknife estimates and 95% confidence limits estimated in Breen et al (2015) are as follows: *P:* 0.84 (0.74, 0.93); *k:* 0.21 h^{-1} (0.13, 0.29 h^{-1})

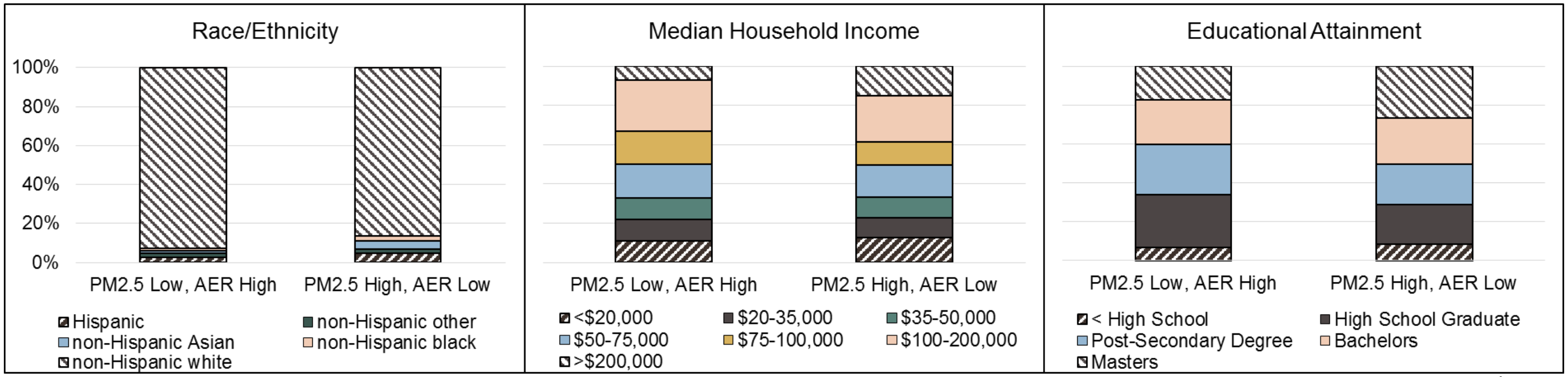

Figure S3.1. Sociodemographic characteristics of block groups containing the residential parcels with the lowest air exchange rates (AER) (=<10th %tile) in areas with the highest ambient $PM_{2.5}$ (=>90th %tile) versus block groups containing parcels with the highest AER and lowest $PM_{2.5}$.
Source: US Census 2010.

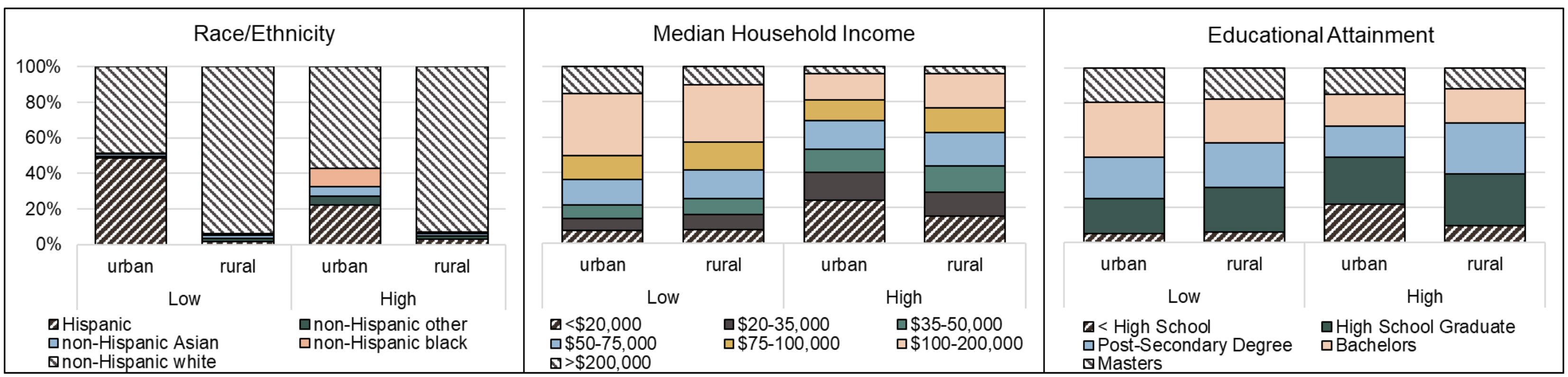

Figure S3.2. Sociodemographic characteristics of block groups containing the residential parcels with the lowest air exchange rates (AER) in areas with the lowest ambient $PM_{2.5}$ (low-exposure, =<10th %tile) versus block groups containing parcels with the highest AER and $PM_{2.5}$ (high-exposure, =>90th %tile), stratified by urban/rural class.
Source: US Census 2010.

CHAPTER 4. PRENATAL AND POSTNATAL AMBIENT AIR POLLUTION EXPOSURE: ASSOCIATION WITH LONGITUDINAL WEIGHT GROWTH TRAJECTORIES IN EARLY CHILDHOOD

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Abstract

Air pollution exposure during pregnancy has been associated with impaired fetal growth and postnatal weight gain throughout the life course. However, there are few studies of longitudinal weight growth trajectories in early childhood to determine the time course of weight gain as a function of air pollution exposures. Using electronic medical record and survey data collected from participants in the ethnically diverse and highly mobile Boston-based Children's HealthWatch cohort (n=4797), we examine the association between $PM_{2.5}$ exposure and residential distance to road and sex-specific weight (kg) growth trajectories from birth to age six. Females exposed to average prenatal $PM_{2.5} > 9.5$ µg/m^3 had significantly higher weights compared to females exposed to <=9.5 µg/m^3 throughout the study period (0.16 kg at 24 months, 0.78 kg at 60 months). Male weights were significantly lower with higher prenatal $PM_{2.5}$ after 24 months of age, with differences increasing with time (-0.17 at 24 months, -0.62 kg at 60 months). This association remained consistent among low birth weight (< 2500 g) females, but did not differ by birth weight status in males. Weights did not differ by categories of distance to road and were not associated with postnatal $PM_{2.5}$ exposure. Our findings demonstrate the complex association between environmental exposures and childhood weight trajectories and emphasize the importance of sex-stratified analyses.

Background

Evidence is accumulating that weight growth trajectories *in utero* and during early postnatal periods are predictive of childhood overweight and obesity (Baird et al., 2005; Stettler et al., 2002). Investigating when the onset of childhood overweight and obesity occurs is of increasing interest to understand the etiology of childhood and adult obesity and to identify critical periods for intervention (Stettler et al., 2002). Early-life overweight and obesity are associated with a range of chronic adverse health outcomes such as type 2 diabetes, coronary heart disease (Barker et al., 2005), and hypertension (Barker et al., 2002; Dennison et al., 2006; Matthews et al., 2017; Reilly et al., 2005; The GBD 2015 Obesity Collaborators, 2017). Although genetic susceptibility to overweight and obesity exists (Giles et al., 2015; Linabery et al., 2013; Parsons et al., 2001), the rapid rise of obesity implicates environmental risk factors as contributors to this trend (Gillman, 2005). In this study, we investigate whether prenatal and early postnatal ambient air pollution exposure is associated with early-childhood growth trajectories.

Exposure to ambient air pollution such as particulate matter is a ubiquitous and modifiable risk factor. Inhalation of particulate matter 2.5 microns or less in aerodynamic diameter ($PM_{2.5}$) during pregnancy can interfere with fetal growth via oxidative stress (OS), intrauterine inflammation, endothelial function and altered mitochondrial function (de melo et al., 2015; Janssen et al., 2016; Kannan et al., 2006). Both animal and human studies have suggested that placental OS in the fetal environment is the most likely biological mechanism connecting prenatal $PM_{2.5}$ and traffic exposure with measures of postnatal weight (Bolton et al., 2012; Sun et al., 2009). OS occurs with excess creation of reactive oxygen species, which signal transcription of genes that are important in maintaining cardiovascular homeostasis subsequent adipogenesis (Thompson and Al-Hasan, 2012). These biological processes may alter trophic mechanisms that control growth through the life course (Barker et al., 2002).

The vast majority of epidemiological and preclinical studies to date associating ambient air pollution and weight have focused almost exclusively on prenatal exposure with weight outcomes (birth weight, raw weight, BMI, adiposity) measured cross-sectionally (Jerrett et al., 2014; Zheng et al., 2016). Modelling weight as a longitudinal outcome - growth trajectories - is a more informative measure than weight modelled as a cross-sectional outcome in understanding steps on the causal pathway between early-life air pollution exposure and morbidities later in life. A limited number of studies have investigated the link between prenatal air pollution exposure and infant and early-childhood growth trajectories (Fleisch et al., 2015; Malmqvist et al., 2017; Mao et al., 2016; McConnell et al., 2015), and none have assessed the link between postnatal exposures and weight growth.

Using electronic medical records (EMR) and surveys administered to obtain detailed maternal and child demographic information, we examined associations of weight growth trajectories from birth to age six years with prenatal and postnatal $PM_{2.5}$ and distance to road (traffic) exposure in the Boston-based Children's HealthWatch (CHW) cohort. Exposure was assessed using concentrations from spatially and temporally resolved $PM_{2.5}$ predictions at 1 km^2 resolution and residential proximity to major roads. Based on previous evidence associating prenatal $PM_{2.5}$ and traffic exposure with low birth weight (LBW), we hypothesize a significant association between prenatal and postnatal $PM_{2.5}$ exposures and weight growth rates in our study population.

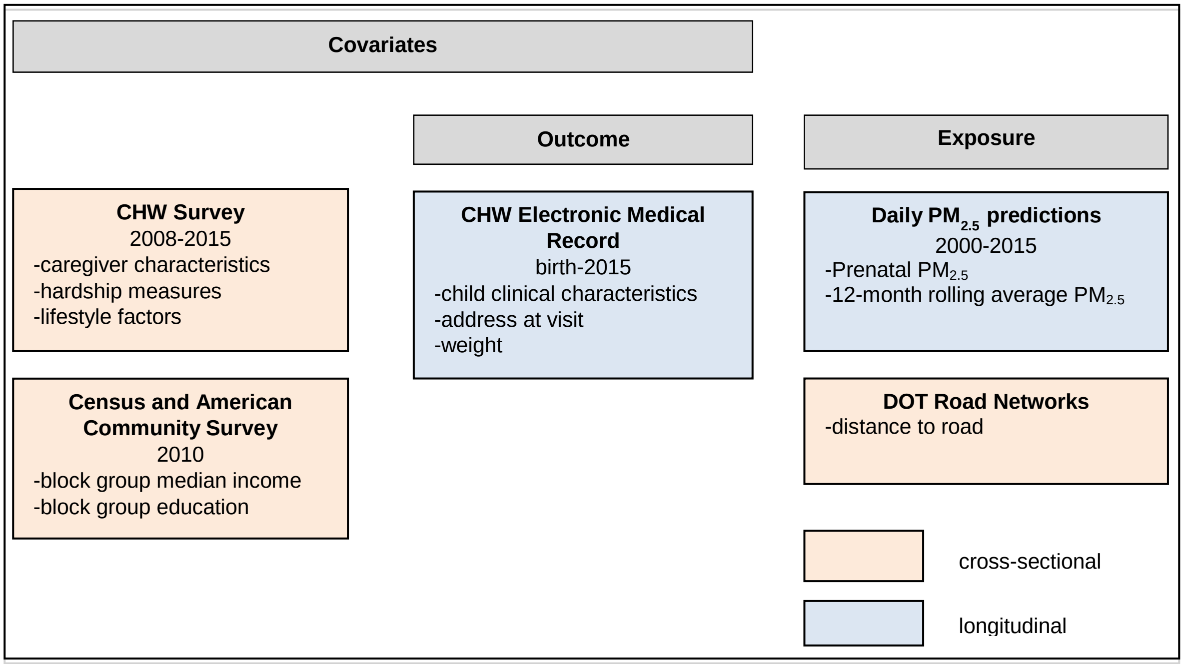


Figure 4.1. Data sources linked to create final analytical dataset for growth trajectory analysis.

Abbreviations: CHW (Children's HealthWatch); DOT (Department of Transportation); EMR (Electronic Medical Record); ACS (American Community Survey); AER (air exchange rate)

Methods

Study Population

The study population was identified from participants who enrolled in CHW in Boston, Massachusetts at the Boston Medical Center between January 1, 2008 and December 31, 2015. CHW is an ongoing, sentinel surveillance study that gathers clinical and interview data from primary care sites or non-urgent emergency department (ED) visits (Cutts et al., 2011). Institutional review board approval was obtained from Boston

Medical Center prior to data collection.

At primary care or ED visits, trained CHW interviewers survey caregivers accompanying children younger than 48 months in a private setting. The survey covers multiple domains, including demographic and socioeconomic characteristics, breastfeeding practices, smoking status, child health status, and information about material hardship – including housing, food and energy insecurity. Respondents were excluded if the interviewee was not the primary caregiver, if they did not speak English or Spanish, were not knowledgeable about the child's household, had been interviewed previously that year, lived out of state, or did not consent to participate. Caregivers of critically ill or injured children were not approached.

EMR covariate and weight data

As shown in Figure 4.1, CHW survey data were linked to EMRs and to multiple spatial exposure databases. CHW surveys were matched to the EMR based on date of child's CHW interview, gender and date of birth. EMR data coverage included birth until December 31st, 2015, resulting in an EMR data range from 2005 to 2015. Birth weight and weight (kg) at each visit were extracted from the EMR, as well as address at each visit, age at each visit (months), gestational age (weeks), visit type (inpatient or outpatient), primary diagnosis for admission (ICD-10 code), child sex, child date of birth, and visit type (inpatient or outpatient). Missing EMR birth weight, gender, and gestational age data were imputed using CHW survey data. Correlation coefficients between the two data sources were 0.97, 0.99 and 0.95 for birth weight, gender, and gestational age, respectively. Child weight (kg) at each visit was measured by medical professionals during ED and primary care visits. We used weights from both inpatient and outpatient visits, and performed a sensitivity analysis excluding inpatient weights.

Analytical Sample Selection

Only participants with two or more weight measurements over the study period were included in the study. We excluded visits with missing weight data, if exact weight measurements were repeated between visits, if an address was missing, could not be geocoded, or Boston Medical Center was listed as the address. Biologically implausible weight values, defined as a sex-specific weight-for-age z score of less than -6 or more than 5 were also dropped from analyses, as recommended by the Centers for Disease Control and Prevention (Centers for Disease Control, 2017). IDs with missing covariate information from the CHW survey or the EMR were additionally excluded. This process yielded a final analysis sample of 4,797 caregiver/child dyads with over 70,649 visits (Figure S4.1).

Exposure Assessment

Geocoding. Address at each medical record visit was geocoded to the parcel, using a reference layer developed through a collaboration between MassGIS, the State 911 Department, and the state's Executive Office of Public Safety and Security (MassGIS, 2017). Of all addresses listed in the EMR, 0.9% were either missing or listed as a P.O. Box. Of the remaining addresses that were included in the geocoding process, 3.6% were unmatched. Geocoded addresses at each visit were linked to predicted $PM_{2.5}$ data and a number of other spatial covariates, discussed in more detail below.

Prenatal and postnatal ambient $PM_{2.5}$. $PM_{2.5}$ at 1 km^2 resolution was obtained from a dataset that has been validated and used in previous studies of air pollution (Fleisch et al., 2016; Mehta et al., 2016; Shi et al., 2016). Details of the $PM_{2.5}$ prediction model can be found in (Kloog et al., 2014). Briefly, this modeling approach used a combination of aerosol optical depth (AOD) satellite data retrieved using the multi-angle implementation of atmospheric correction (MAIAC) algorithm, land use, and meteorological variables and outdoor monitor $PM_{2.5}$ concentrations to calculate daily $PM_{2.5}$ predictions on a 1 km^2 grid between 2000 and 2015 (Kloog et al., 2014).

We assigned $PM_{2.5}$ to the geocoded addresses using the closest 1 km^2 grid centroid. We calculated average $PM_{2.5}$ concentration over the prenatal period using the address, date of birth and gestational age (in weeks). We categorized prenatal $PM_{2.5}$ as a bivariate above and below the median (9.5 $\mu g/m^3$), and tested $PM_{2.5}$ categorized as tertiles. For postnatal exposure, we computed the 12-month moving average of $PM_{2.5}$ concentrations preceding and including the date of each hospital visit recorded in the EMR. This approach allows us to incorporate residential address moves into the exposure estimate. If address moves occurred during periods longer than the rolling average period, we assumed that the subject moved on the date halfway between the two visits. Because postnatal $PM_{2.5}$ is time-varying, we included it in the model as a continuous variable to examine change in slope per 10 $\mu g/m^3$ increase in $PM_{2.5}$, as has been modelled previously (Eze et al., 2015; Malmqvist et al., 2017; Shi et al., 2016).

Distance to Road. We used the Massachusetts Department of Transportation Roads layer to calculate the Euclidian distance between each geocoded residential address and the nearest street segment defined in classes 1 through 4 (MassGIS, 2014). We tested linear distance to road, but ultimately categorized as <50 meters, 50-200 meters and >200 meters on the basis of previous health studies and to account for exponential decay in traffic pollutants with distance from source (Jerrett et al., 2014; Lebret et al., 2000; Zhu et al., 2002).

Covariates. We assigned block group-level covariates from Census 2010 and ACS 2006-2010 5-year summary data to each geocoded address. Linked covariates included median block group household income and percent with less than a high school degree.

Individual covariates tested for model inclusion derived from the EMR include: number of moves within the study period (continuous), child's birth weight (binomial categorized at >2500 grams), child's gestational age (continuous and binomial categorized at >37 weeks), child sex, and birth date. From ACS 2006-2010 we tested median block group household income (continuous) and percent with less than a high school degree (continuous).

From the CHW survey we ascertained year of enrollment, caregiver BMI (underweight, normal weight, overweight, obese), caregiver race/ethnicity (non-Hispanic white, non-Hispanic black, Hispanic, other), breastfed during pregnancy (yes/no), caregiver smoking status in the past five years (yes/no), caregiver immigration status (U.S. born, yes/no) and caregiver educational attainment (no schooling or some high school, high school, postsecondary), a composite measure of food, energy and housing insecurity (referred to herein as "cumulative hardship") (Frank et al., 2010), and mother's age at birth (derived from child date of birth and mother's age at CHW enrollment date).

Weight growth model

To estimate the association between air pollution exposures (i.e. average prenatal $PM_{2.5}$ exposure, distance to road and postnatal $PM_{2.5}$) and growth trajectories, we used a two stage modeling approach.

Stage 1: Modelling Weight for Age Trajectories. We applied generalized additive mixed models to assess sex-specific postnatal growth trajectories. The models included a random intercept for child and a random slope for age to account for repeated measurements within subject and to allow for heterogeneity in trends over time (Fitzmaurice et al., 2011; Howe et al., 2016). This approach allows for correlated repeated weight measurements and varying number of measures per child. We built two forms of multi-level models to approximate growth trajectories separately for non-time-varying and time-varying exposure: cubic polynomial splines for prenatal $PM_{2.5}$ and distance to road exposures (non-time-varying) and piecewise linear splines for postnatal $PM_{2.5}$, a time-varying exposure. Linear spline models with cubic polynomial terms allow for a close approximation of the true growth function, but the coefficients are not interpretable. Piecewise linear spline models are not biologically plausible because the shape of the trajectory assumes linear slopes between knot points. However, the coefficients of a piecewise linear model are easily interpretable. Both modelling approaches have been shown to produce good model fit in this and several other cohorts (Chirwa et al., 2014; Grajeda et al., 2016; Linabery et al., 2013; Lourenço et al., 2012;

O'Keeffe et al., 2015; Patel et al., 2014; Tilling et al., 2014).

We used an iterative process to test combinations of one, two, three or four knot points at knot placements 3, 6, 9, 12, 18, 24, 36, and 40 months in both cubic polynomial and piecewise linear models. Two and three degree polynomial functions were explored by adding linear, quadratic, and cubic terms to the model at the aforementioned knot points. We assessed model fit by comparing Akaike Information Criterion (AIC) and log-likelihood values between non-nested and nested models, respectively. The best fit polynomial model for males and females included quadratic terms at 6 and 12 months. The best fit piecewise linear spline model included knots at 3, 6, 12 and 24 months for males, and 3, 6, 12 and 18 months for females.

Stage 2: Multi-variable Model Building. In the second step, we tested several covariates *a priori* known to be conceptually related to childhood postnatal weight, $PM_{2.5}$ and traffic exposure. Using a likelihood ratio test, we found that including interactions between covariates and age terms in the model provided a significantly better fit than simply including the covariates as main effects, suggesting that the effect of these predictors varies over age.

We included the following covariates in the final model: caregiver race/ethnicity, cumulative hardship (categorical), child's gestational age (categorical), block group median household income (continuous), and caregiver immigration status. After developing a final covariate-only model, we added the main exposures (prenatal $PM_{2.5}$, distance to road, and 12 month moving-average postnatal $PM_{2.5}$ exposure) as main effects and as interactions with age terms to allow the shape of the curve to differ between groups. To maintain consistency, all exposure models included the same set of covariates.

This approach yielded three separate models:

- Model 1: Cubic polynomial spline multi-level model, average prenatal $PM_{2.5}$ categories
- Model 2: Cubic polynomial spline multi-level model, average postnatal distance to highway categories
- Model 3: Linear spline multi-level mode, 12-month rolling average postnatal $PM_{2.5}$ (continuous)

Models 1 and 2 took the form:

Males and Females:

$Y_{ij}=B_0+B_1age_{ij}+ B_2age_{ij}^2+ B_3age_{ij}^3+ B_4(age_{ij}\text{-6months})^2+ B_5(age_{ij}\text{-12months})^2+B_6exposure_i + B_{7\text{-}n}covariates_i+(B_nage_{ij}*(exposure_i+ covariates_i))+(B_nage_{ij}^2*(exposure_i+ covariates_i))+ (B_nage_{ij}^3*(exposure_i+ covariates_i))+ ((B_n(age_{ij}\text{-6months})^2)*(exposure_i+ covariates_i))+ (B_n(age_{ij}\text{-12months})^2*(exposure_i+ covariates_i))+b_{0i}+b_{1i}age_{ij}+e^{ij}$

and Model 3 took the form:

Males:
$Y_{ij}=B_0+B_1age_{ij}+ B_2(age_{ij}\text{-3months})+ B_3(age_{ij}\text{-6months})+ B_4(age_{ij}\text{-12months})+ B_5(age_{ij}\text{-24months})+ B_6exposure_i + B_{7\text{-}n}covariates_i+(B_nage_{ij}*(exposure_i+ covariates_i))+(B_n(age_{ij}\text{-3months})*(exposure_i+ covariates_i))+ (B_n(age_{ij}\text{-6months})*(exposure_i+ covariates_i))+ ((B_n(age_{ij}\text{-12months})*(exposure_i+ covariates_i))+ (B_n(age_{ij}\text{-24months})*(exposure_i+ covariates_i))+b_{0i}+b_{1i}age_{ij}+e_{ij}$

Females:
$Y_{ij}=B_0+B_1age_{ij}+ B_2(age_{ij}\text{-3months})+ B_3(age_{ij}\text{-6months})+ B_4(age_{ij}\text{-12months}) + B_5(age_{ij}\text{-18months}) + B_6exposure_i + B_{7\text{-}n}covariates_i+(B_nage_{ij}*(exposure_i+ covariates_i))+(B_n(age_{ij}\text{-3months})*(exposure_i+ covariates_i))+ (B_n(age_{ij}\text{-6months})*(exposure_i+ covariates_i))+ ((B_n(age_{ij}\text{-12months})*(exposure_i+ covariates_i))+ (B_n(age_{ij}\text{-18months})*(exposure_i+ covariates_i))+b_{0i}+b_{1i}age_{ij}+e_{ij}$

where $exposure_i$ is average prenatal $PM_{2.5}$, distance to road, and postnatal rolling-average $PM_{2.5}$ in Models 1, 2, and 3 for the i^{th} subject, respectively; $covariates_i$ are non-time-varying covariates for the i^{th} subject; $b_{0i}+b_{1i}$ are subject specific random intercept and slope for the i^{th} subject.

To optimize interpretability of Models 1 and 2, we use model predictions to estimate differences in weight between levels of prenatal $PM_{2.5}$ and near roadway pollution over the weight trajectory at specified ages. For Model 3, we assess change in growth rates (slope in kg per month) for every 10 $\mu g/m^3$ increase in $PM_{2.5}$ exposure within the age ranges defined by the knot points.

Secondary Analyses.

We performed a sub-analysis including mother's BMI (underweight, normal, overweight, obese) at study enrollment as a model covariate given literature showing that maternal and paternal weight are associated with childhood growth and obesity outcomes (Giles et al., 2015; Linabery et al., 2013; Parsons et al., 2001). This analysis was conducted with a subset of the study population (63%) as the remaining participants were missing biological mother's BMI.

We tested effect measure modification (EMM) by stratifying by levels of multiple potential modifiers. We tested EMM by birth weight (<2500 grams), as growth trajectories may differ by birth weight. The phenotype of low birthweight followed by catchup growth has been associated with a range of cardiometabolic outcomes (Hales and Barker, 2001; UNICEF and WHO, 2004; Vaag et al., 2012). We examined EMM by residential air exchange rate (AER) for the subset of participants for which this variable was estimated. AER may modify residential exposures to $PM_{2.5}$ concentration of ambient origin (Meng et al., 2005). We estimated AER using the LBL residential air leakage model as described in (Breen et al., 2015, 2010; Sarnat et al., 2013) and in Chapter 3.

We also performed a sensitivity analysis excluding all subjects born <37 weeks gestation and a separate analysis excluding inpatient weights (Reddy et al., 2011).

We used a p-value of < 0.05 to denote statistical significance in exposure models. All statistical analyses were conducted using R version 3.3, with the software package lme version 3.3 (R Foundation for Statistical Computing, Vienna, Austria).

Results

Population characteristics

Characteristics of the study population stratified by child sex are presented in Table 4.1. The CHW cohort is an ethnically diverse, relatively mobile and low-income population. Cohort participants were 50% non-Hispanic black, 43% have a post-secondary degree, 42% of mothers enrolled are immigrants to the United States and 42% moved at least once during the study period. The average block group median income is $43,871 and $43,697 for males and females respectively, which is lower than the state average block group median household income of $70,114. The mean mother's age at delivery is 27 (SD: 6.3).

Compared to participants who had missing or unmatched addresses, those with geocoded addresses were more likely to have smoked in the five years before CHW enrollment (31% versus 26%) (χ^2=0.05) There were no significant differences between the two groups in immigration status, breastfeeding status, marital status, ethnicity, educational attainment, sex proportions, gestational age, cumulative hardship, insurance status, mother's BMI at enrollment, and small for gestational age proportions.

The median distance to a major road was 82.7 (IQR: 37.6-177.5) and 82.0 (IQR: 39.6-169.5) meters for males and females, respectively. Mean prenatal and postnatal $PM_{2.5}$ concentrations were similar between males 9.6 (SD:1.1, range: 6.5-14.0) $\mu g/m^3$ and females 9.6 (SD:1.2, range: 6.3-14.1) $\mu g/m^3$. Male and female 12-month rolling average $PM_{2.5}$ concentrations were 8.8 $\mu g/m^3$ (SD: 1.5, range: 1.4-44.4) and 8.9 $\mu g/m^3$ (SD:1.5, range: 1.4-44.4), respectively.

Table 4.1. Study Population Characteristics

	Males		Females	
	n	%	n	%
Child Characteristics				
Total	2582		2215	
Birth weight (g) (% missing: 1.6 males, 1.1 females)				
>=2500	2250	88.6	1900	86.7
<2500	290	11.4	291	13.3
Gestational age (% missing: 0.4 males, 0.4 females)				
>=37 weeks	2164	84.2	1893	85.8
<37 weeks	407	15.8	313	14.2
Breastfed During Pregnancy (% missing: 0.8 males, 0.6 females)				
Yes	1985	77.5	1702	77.3
No	577	22.5	499	22.7
Cumulative hardship (% missing: 12.7 males, 11.6 females)[a]				
0 hardships	797	35.3	664	33.9
1-3 hardships	1280	56.8	1129	57.7
>3 hardships	178	7.9	164	8.4
Number of overall visits (inpatient and outpatient)				
mean (SD)	14.4	14.4	14	13.4
Block group median income ($)				
mean (SD)	43871	21789.1	43697.2	22126.3

Self-reported caregiver characteristics				
Marital status (% missing: 0.5 females, 0.4 females)				
married	928	36.1	787	35.7
not married	1641	63.9	1419	64.3
Ethnicity (% missing: 1.2 males, 1.0 females)				
Hispanic	909	35.6	776	35.4
Black, Non-Hispanic	1285	50.4	1108	50.5
White, Non-Hispanic	221	8.7	192	8.8
Other	136	5.3	117	5.3
Education (% missing: 0.5 males, 0.3 females)				
less than high school	604	23.5	531	24.0
high school graduate	844	32.9	732	33.1
post-secondary	1120	43.6	946	42.8
Country of birth[b] (% missing: 0.8 males, 0.3 females)				
U.S. born	1475	57.6	1275	57.7
not U.S. born	1087	42.4	933	42.3
Smoked in last 5 years (% missing: 4.1 males, 2.9 females)				
yes	616	24.9	564	26.2
no	1860	75.1	1587	73.8
Age at child's birth				
mean (SD)	26.8	6.3	27	6.3

[a]Refers specifically to biologic mother
[b]Score derived from questions about housing, energy and food hardship
SD = standard deviation

Estimated Weight

Figure S4.2 displays adjusted generalized additive mixed models of growth rates by sex. As expected, both males and females have an exponential rate of growth in the first few months of life, which slows and becomes linear around 12 months of age. Table S4.1 displays predicted growth rates. Males have higher rates of growth from 0-3 months of age compared to females, and growth rates for both sexes level off after 12 months of age, as demonstrated in Figure S4.2.

Table 4.2. Observed and Predicted Weights (kg) in Study Population compared to General U.S. Population Growth Standards

	Male				Female			
Age (months)	Observed[a]	Cubic model prediction	Linear model prediction	U.S. Population[b]	Observed[a]	Cubic model prediction	Linear model prediction	U.S. Population.[b]
0	3.3	2.9	3.1	3.4	3.2	3.1	3.0	3.2
3	6.1	6.1	6.2	6.4	5.4	5.6	5.6	5.8
6	8.0	8.0	8.1	7.9	7.3	7.3	7.5	7.3
12	10.0	10.1	10.3	9.6	9.3	9.0	9.6	8.9
18	11.6	11.5	11.8	10.9	11.0	10.2	11.1	10.2
24	13.0	12.9	13.3	12.2	12.4	11.3	12.5	11.5
36	15.4	15.4	15.9	14.3	14.6	13.4	15.3	13.8
48	17.9	18.0	18.4	16.3	17.7	15.6	18.0	15.9
60	20.4	20.5	21.0	18.5	20.1	17.7	20.8	18.0
72	21.9	23.0	24.1	20.5	22.2	19.8	24.0	20.3

[a]median observed weight from EMR within 2 weeks of listed age, unadjusted
[b]U.S. Population weights derived from WHO growth standards for ages 0-2 years, and CDC growth standards for ages 2-6 years (Centers for Disease Control, 2017)

Table 4.2 presents weights estimated using both the cubic polynomial and piecewise linear spline compared to U.S. reference population values and observed average weight in the CHW database at ages 0 to 72 months. Overall, models produced values close to observed weights. Cubic polynomial models slightly underestimated birth weight and slightly overestimate weight after 60 months for males, and slightly underestimated weights after 12 months for females. Piecewise linear models slightly overestimated weights for both males and females at all ages. Observed weights in the study population were lower than the U.S. population during the early infancy period (0-3 months), but were higher at all other ages.

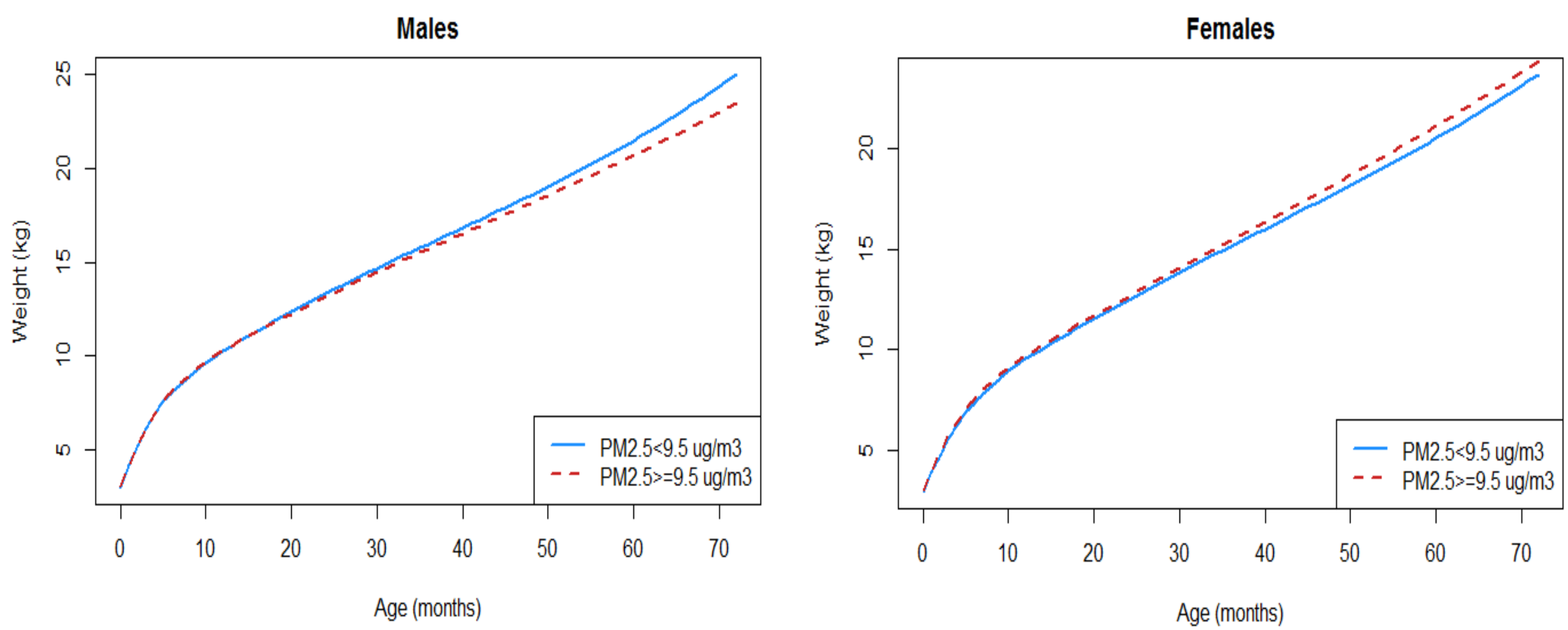


Figure 4.2. Predicted weight (kg) over age (months) by levels of average prenatal $PM_{2.5}$
Models adjusted for: age, age^2, age^3, quadratic spline terms at 6 and 12 months, gestational age, ethnicity, education, immigrant status, cumulative hardship and block group median income

Table 4.3. Mean Predicted Weight (kg) by Prenatal $PM_{2.5}$ Category

Age	Birth	3 months	6 months	12 months	18 months	24 months	36 months	48 months	60 months	72 months
Males (n=2213, 29,843 weight measurements)										
<9.5 μg/m^{3}[a]	3.00	6.16	8.09	10.25	11.87	13.33	15.99	18.58	21.48	25.06
	(2.94, 3.06)	(6.10, 6.22)	(8.03, 8.15)	(10.18, 10.33)	(11.78, 11.96)	(13.22, 13.45)	(15.82, 16.15)	(18.36, 18.81)	(21.20, 21.77)	(24.68, 25.44)
>=9.5 μg/m^{3}[a]	3.03	6.17	8.14	10.30	11.78	13.17	15.73	18.17	20.70	23.52
	(2.96, 3.10)	(6.11, 6.24)	(8.08, 8.21)	(10.22, 10.38)	(11.69, 11.88)	(13.05, 13.29)	(15.56, 15.90)	(17.95, 18.40)	(20.42, 20.98)	(23.17, 23.87)
Absolute Δ (kg)	-0.03	-0.01	-0.05	-0.04	0.09	0.16	0.26	0.41	0.78	1.54
p-value [b]	0.53	0.79	0.25	0.44	0.20	0.05	0.03	0.01	0.0001	< .00001
Females (n=1866, 24,279 weight measurements)										
<9.5 μg/m^{3}[a]	2.94	5.57	7.35	9.52	11.05	12.47	15.13	17.73	20.50	23.68
	(2.87, 3.00)	(5.50, 5.63)	(7.29, 7.42)	(9.44, 9.60)	(10.94, 11.15)	(12.34 ,12.60)	(14.94, 15.32)	(17.47, 17.99)	(20.17, 20.83)	(23.23, 24.13)
>=9.5 μg/m^{3}[a]	2.97	5.70	7.55	9.71	11.20	12.64	15.42	18.19	21.12	24.36
	(2.90, 3.04)	(5.63, 5.77)	(7.48, 7.62)	(9.62, 9.79)	(11.10, 11.30)	(12.51 ,12.77)	(15.23, 15.60)	(17.95, 18.44)	(20.82, 21.43)	(23.97, 24.74)
Absolute Δ (kg)	-0.03	-0.14	-0.19	-0.19	-0.15	-0.17	-0.28	-0.47	-0.62	-0.68
p-value [b]	0.50	0.01	0.00008	.001	0.03	.077	.035	.011	0.01	0.03

Models adjusted for: age, age^2, age^3, quadratic spline terms at 6 and 12 months, gestational age, ethnicity, education, immigrant status, cumulative risk and block group median income

[a]Values are mean estimated weights in kg (95% CIs)

[b]*p*-values for difference between low and high exposure categories

Prenatal Ambient $PM_{2.5}$ Exposure and Longitudinal Childhood Weight Trajectories

All prenatal $PM_{2.5}$ models included age modelled as a cubic polynomial, with quadratic terms at 6 and 12 months of age. The relationship between age and weight is complex and was best captured with multiple polynomial terms, which yielded the best fit but were not directly interpretable.

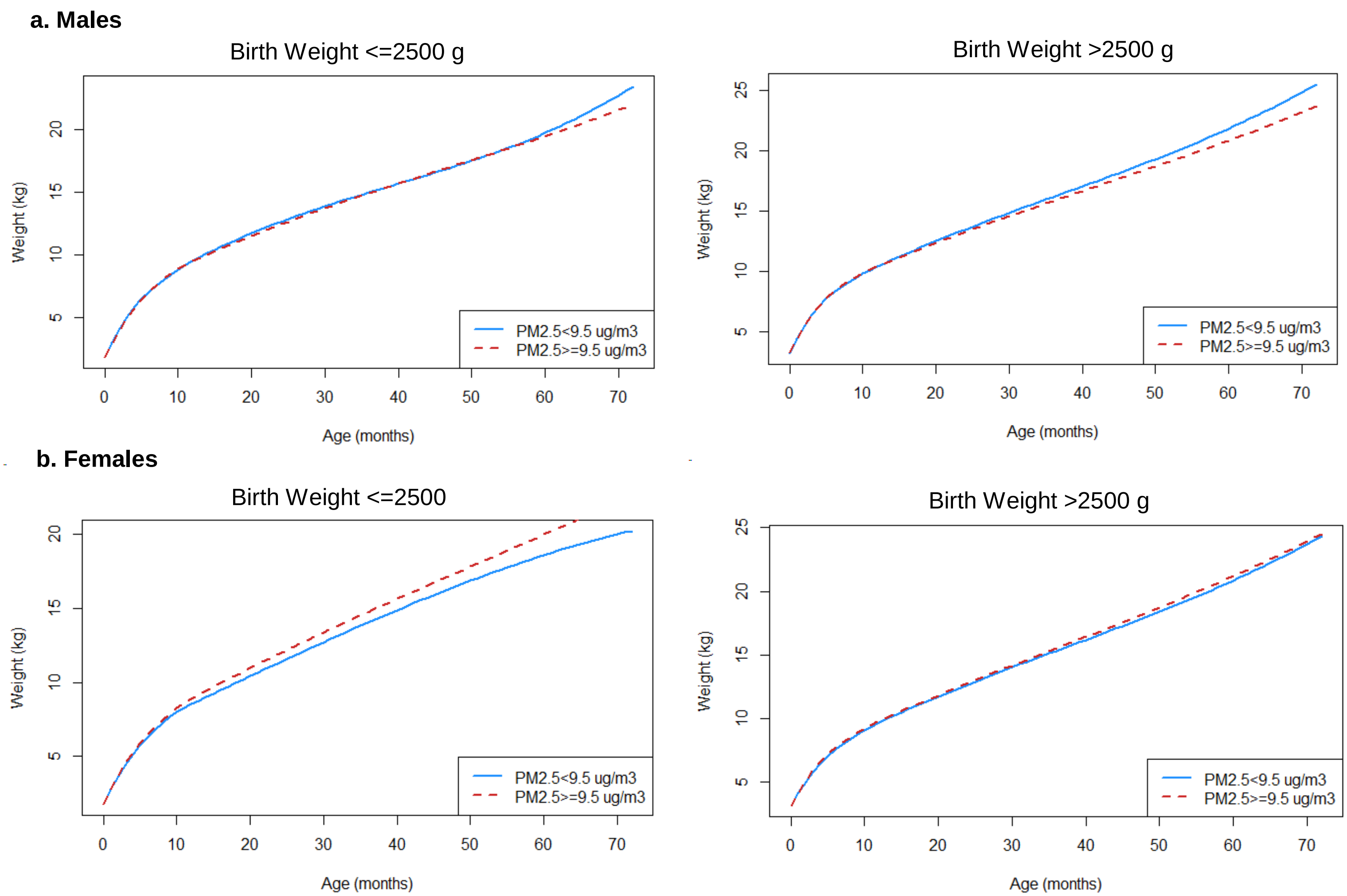
Figure 4.2 shows estimated childhood weight trajectories by levels of $PM_{2.5}$ below the median (solid line) and above the median (dashed line) from birth through 72 months. Among males, above-median prenatal $PM_{2.5}$ exposure results in growth trajectories that are significantly lower compared to the below-median prenatal $PM_{2.5}$ exposure group from 2-6 years of age. The model predicts a 0.16 kg lower weight at 24 months and 1.54 kg lower weight at 72 months. We see the opposite effect among females, where above-median prenatal $PM_{2.5}$ is associated with significantly higher weights at all ages, with the exception of birth weight. The greatest difference in weight was at 72 months, where high exposure groups had weights 0.68 kg higher than low exposure groups (Table 4.3).

Mother's BMI at enrollment was significantly associated with weight across the growth trajectory in all male prenatal $PM_{2.5}$ models, but not in female $PM_{2.5}$ models. When included in both models, the direction and magnitude of effects were similar to Model 1. When restricted to full-term births (>37 weeks) among males, the direction of effect at all ages was similar across the full trajectory, compared to Model 1 not restricted to full-term births. We found similar results when we categorized $PM_{2.5}$ into tertiles: with similar growth curves between the 50th and 75th percentiles and lower growth curves for those in the 25th percentile (results not shown).

Impact of Birth Weight

Results for polynomial age models for prenatal $PM_{2.5}$, stratified by low birth weight (LBW, <2500 g) and non-low birth weight (>=2500 g), are presented in Figures 4.3a and 4.3b, and Tables 4.4a and 4.4b.

As with the unstratified sample, estimated weights were significantly higher for the lower prenatal $PM_{2.5}$ group in both birth weight strata among males, after 24 months. Absolute weight differences were slightly more pronounced in the non-LBW group as compared to the LBW group (e.g. 0.99 kg in non-LBW vs. 0.85 kg in LBW at 60 months). Among females, differences between high and low $PM_{2.5}$ exposure groups were greater among LBW compared to non-LBW females. The above-median prenatal $PM_{2.5}$ group had 0.24 kg greater weight at 6 months of age and 3.28 kg greater weight at 72 months of age in LBW females.



Figures 4.3a and b. Predicted weight (kg) over age (months) by levels of average prenatal $PM_{2.5}$, stratified by birth weight categories Models adjusted for: age, age^2, age^3, quadratic spline terms at 6 and 12 months, gestational age, ethnicity, education, immigrant status, cumulative risk and block group median income

Table 4.4a. Estimated weight (kg) by $PM_{2.5}$ Categories, Males, Stratified by Birth Weight

	Birth	3 months	6 month	12 months	18 months	24 months	36 months	48 months	60 months	72 months
>2500 g, (1986 IDs, 25064 measures)										
<9.5 μg/m^3 [a]	3.20	6.37	8.28	10.39	11.98	13.45	16.16	18.83	21.83	25.52
	(3.13, 3.26)	(6.31, 6.43)	(8.22, 8.34)	(10.31, 10.46)	(11.89, 12.08)	(13.33, 13.57)	(15.98, 16.34)	(18.58, 19.07)	(21.52, 22.14)	(25.10, 25.94)
>=9.5 μg/m^3 [a]	3.22	6.38	8.32	10.41	11.88	13.26	15.82	18.28	20.83	23.66
	(3.15, 3.29)	(6.31, 6.45)	(8.25, 8.39)	(10.33, 10.49)	(11.78, 11.98)	(13.13, 13.38)	(15.63, 16.01)	(18.03, 18.53)	(20.52, 21.15)	(23.26, 24.05)
Δ	-0.02	-0.01	-0.04	-0.02	0.11	0.21	0.34	0.54	0.99	1.86
p-value [b]	0.63	0.88	0.37	0.68	0.13	0.02	0.01	0.002	0.00001	< .00001
<=2500 g (245 IDs, 4779 measures)										
<9.5 μg/m^3 [a]	1.87	5.02	7.07	9.49	11.17	12.62	16.13	18.78	21.75	25.36
	(1.67, 2.06)	(4.85, 5.18)	(6.90, 7.24)	(9.29, 9.69)	(10.92, 11.42)	(12.30, 12.93)	(15.96, 16.31)	(18.55, 19.02)	(21.45, 22.05)	(24.96, 25.76)
>=9.5 μg/m^3 [a]	1.84	4.92	7.03	9.48	11.03	12.43	15.83	18.32	20.89	23.75
	(1.62, 2.05)	(4.72, 5.11)	(6.84, 7.23)	(9.26, 9.71)	(10.76, 11.30)	(12.10, 12.76)	(15.65, 16.01)	(18.08, 18.56)	(20.59, 21.19)	(23.36, 24.13)
Δ	0.03	0.10	0.04	0.01	0.14	0.19	0.30	0.47	0.85	1.62
p-value [b]	0.84	0.44	.44	.97	0.45	0.04	0.02	.007	0.0001	< .00001

Models adjusted for: age, age^2, age^3, quadratic spline terms at 6 and 12 months, gestational age, ethnicity, education, immigrant status, cumulative risk and block group median income

Δ=absolute difference in weight (kg) between low and high exposure categories

[a]Values are mean estimated weights in kg (95% CIs)

[b]*P*-values for difference between low and high exposure categories

	Birth	3 months	6 months	12 months	18 months	24 months	36 months	48 months	60 months	72 months
>2500 g, (1625 IDs, 20521 measures)										
<9.5 μg/m^{3a}	3.14 (3.08, 3.21)	5.08 (5.02, 5.15)	7.54 (7.47, 7.61)	9.68 (9.60, 9.77)	11.24 (11.13, 11.35)	12.67 (12.53, 12.81)	15.32 (15.11, 15.52)	17.93 (17.65, 18.21)	20.83 (20.47, 21.18)	24.32 (23.83, 24.81)
>=9.5 μg/m^{3a}	3.15 (3.08, 3.22)	5.78 (5.71, 5.85)	7.70 (7.63, 7.77)	9.81 (9.73, 9.90)	11.32 (11.22, 11.43)	12.76 (12.62, 12.90)	15.51 (15.31, 15.71)	18.25 (17.99, 18.51)	21.19 (20.86, 21.51)	24.50 (24.09, 24.91)
Δ	-0.01	-0.11	-0.16	-0.13	-0.09	-0.09	-0.19	-0.32	-0.36	-0.18
p-value[b]	0.88	0.03	0.002	0.25	0.25	0.36	0.18	0.10	-0.14	0.58
<=2500 g (241 IDs, 3758 measures)										
<9.5 μg/m^{3a}	1.78 (1.59, 1.97)	4.44 (4.28, 4.60)	6.34 (6.18, 6.50)	8.58 (8.39, 8.77)	10.01 (9.76, 10.26)	11.39 (11.06, 11.71)	13.96 (13.47, 14.45)	16.27 (15.59, 16.95)	18.27 (17.39, 19.15)	19.92 (18.68, 21.16)
>=9.5 μg/m^{3a}	1.81 (1.58, 2.04)	4.52 (4.33, 4.71)	6.58 (6.40, 6.77)	9.00 (8.78, 9.21)	10.39 (10.12, 10.67)	11.85 (11.50, 12.21)	14.88 (14.35, 15.41)	17.90 (17.18, 18.61)	20.73 (19.81, 21.64)	23.19 (22.00, 24.38)
Δ	-0.03	-0.08	-0.24	-0.42	-0.38	-0.47	-0.92	-1.62	-2.46	-3.28
p-value[b]	0.87	0.54	0.05	0.01	0.04	0.06	0.01	0.001	0.0002	0.0002

Table 4.4b. Estimated weight (kg) and 95% CI by $PM_{2.5}$ Categories, Females, Stratified by Birth Weight

Models adjusted for: age, age^2, age^3, quadratic spline terms at 6 and 12 months, gestational age, ethnicity, education, immigrant status, cumulative risk and block group median income

Δ=absolute difference in weight between low and high exposure categories

[a]Values are mean estimated weights in kg (95% CIs)

[b]*P*-values for difference between low and high exposure categories

Impact of residential air exchange rate

We performed a subanalysis on the subset of the population with modelled air exchange rate described in Chapter 3. There was a significant interaction of AER with prenatal $PM_{2.5}$ among females ($p=0.0019$) but not males ($p=0.94$). Stratifying by AER at the median (0.87 air changes per hour), the effect of $PM_{2.5}$ was stronger in the high AER stratum, consistent with increased personal exposure (Figure S4.3). Overall, the absolute difference in weight between $PM_{2.5}$ levels in the high AER group was approximately twice that of the low AER group from 6 to 48 months, with the high $PM_{2.5}$ group consistently having higher weights in both AER strata.

Distance to Road and Longitudinal Childhood Weight Trajectories

Male and female weights were not significantly different among categories of the distance to road metric (results presented in Figure S4.4).

Postnatal 12-Month Average $PM_{2.5}$ Exposure and Weight Trajectories

Table 4.4 shows the association between weight growth (kg/month) and time varying post-natal 12-month moving average of $PM_{2.5}$. From birth to three months of age, we found a weight growth of -0.010 kg/month (95% CI: -0.02, 0.0003) for males and of -0.011 (-0.021, -0.001) kg/month for females per 10 $\mu g/m^3$ increase in $PM_{2.5}$. The association weakened for both males and females over age ranges.

Table 4.4. Growth Rate (kg/mo) per 10 μg/m^3 increase in the 12 month moving average of $PM_{2.5}$ at Different Age Ranges, by Sex

	Males (n=2213)		Females (n=1866)	
	Est.	95% CI	Est.	95% CI
0-3 months	-0.01	(-0.020, -0.0003)	-0.011[a]	(-0.021, -0.001)
3-6 months	-0.008	(-0.030, 0.013)	0.002	(-0.023, 0.028)
6-12 months	0.001	(-0.018, 0.021)	-0.004	(-0.027, 0.020)
12-24 months, males 12-18 months, females	-0.007	(-0.017, 0.003)	0.006	(-0.009, 0.022)
24-72 months, males 18-72 months, females	0.005	(-0.001, 0.011)	-0.001	(-0.010, 0.009)

[a] p-value<0.05
Models adjusted for: age terms, gestational age, ethnicity, education, immigrant status, cumulative risk and block group median income

Discussion

We found a significant association between prenatal $PM_{2.5}$ exposure and sex-specific childhood growth trajectories in a racially/ethnically diverse and highly mobile population. Males exposed to $PM_{2.5}$ greater than 9.5 μg/m^3 had lower weights after 24 months of age compared to low-exposed groups. In contrast, over the growth trajectory, female weights were higher for higher $PM_{2.5}$ prenatal exposure, which was driven by LBW females. Postnatal 12-month rolling average $PM_{2.5}$ was associated only with a decreased growth rate in females from 0-3 months of age. Distance to road metrics were not associated with weight trajectories in childhood.

The existence of sex-specific differences in the association between prenatal $PM_{2.5}$ and growth trajectories is consistent with the broader literature on air pollution and birth outcomes, albeit with considerable variation in the magnitude and direction of effect. The literature on differential measures of weight by sex is mixed, though overall, more studies have reported increased susceptibility to *in-utero* $PM_{2.5}$ exposure among males compared to females (Ebisu and Bell, 2012; Ghosh et al., 2007; Jedrychowski et al., 2009; Lakshmanan et al., 2015). In a systematic review, females were more commonly found to be at higher risk of LBW, but in a re-analysis of data from four reviewed studies, males were at higher risk of LBW in the presence of high prenatal $PM_{2.5}$ (Ghosh et al., 2007). Ebisu and Bell (2012) reported a 3.2% (95% CI: 0.8, 5.6%) lower relative risk of LBW per IQR increase of $PM_{2.5}$ elemental carbon in females compared to males (Ebisu and Bell, 2012). The opposite effect was found in a pregnancy cohort located in Krakow: males had 188.6 g lower birth weight in the 4th compared to 1st quartile of prenatal $PM_{2.5}$ (Jedrychowski et al., 2009).

The literature is sparse with reference to prenatal and postnatal outdoor ambient air pollution exposure studies and sex-specific differences in measures of weight later in life. Chiu et al. (2017) found that 1 $\mu g/m^3$ increase in prenatal-average $PM_{2.5}$ was associated with a 0.36 kg (95% CI: 0.12-0.68) increase in fat mass for males, but not females, and an increase in waist to hip ratio in females at four years of age (Chiu et al., 2017). Animal and human studies have demonstrated that prenatal $PM_{2.5}$ exposure can induce sex-specific epigenetic modifications in leptin methylation, which is associated with adult metabolic disorders (Calderón-Garcidueñas et al., 2014; Chen et al., 2017). Sex-specific differences in energy metabolism and increased OS vulnerability in males have been found in animal studies, and may explain sex-specific differences found here (Mauvais-Jarvis, 2015).

Considering other environmental exposures, Mora et al. (2016) found similar sex-specific effects to those found here, with consistent positive associations between prenatal plasma PFAS and BMI as well as physiological measures of adiposity only later in childhood, and the effect was less pronounced in males compared to females. Sex-specific effects were also found with prenatal BPA exposure consistently positively associated with BMI z-score, waist circumference and skinfold thicknesses in females and, like our study, finding an opposite direction of effect among males, though estimates did not reach significance (Yang et al., 2017). Though associations with BPA, phthalates and PFAS analytes are more likely a consequence of endocrine processes, sex-specific effects on placental epigenetic processes may be shared by $PM_{2.5}$ and other exposures of interest. Further studies are needed to elucidate sex-specific effects of $PM_{2.5}$ on weight gain in different populations and that consider the mediating or modifying effects of other potential risk factors not tested in this study.

Beyond the sex-specific effects, our findings are broadly consistent with a growing literature linking air pollution exposures with childhood growth. In a Boston-area pregnancy cohort, Fleisch et al. (2015) found increased odds of weight-for-length >95th percentile at 6 months of age in 4th quartile third-trimester $PM_{2.5}$ (β =-0.08 units, 95% CI: -0.2, 0.04) and distance to roadway <50 compared to the referent group, though estimates were not statistically significant. Though these findings with reference to distance to roadway are not in line with ours, the phenotype of low-birth weight followed by rapid and sustained postnatal weight gain with higher compared to lower exposures support our findings in females. In a follow-up study to Fleisch et al. (2015), children whose mothers lived <50 m from a major roadway at the time of delivery had 2.1 kg (95% CI: 0.8, 3.5) greater total fat mass compared to children (median 7.7 years of age) living >=200 m (Fleisch et al., 2016). Inconsistent with our findings, this same study found each IQR increase in one-year average $PM_{2.5}$ concentrations prior to each measurement occasion was associated with lower BMI-z score, total and truncal fat mass in mid-childhood (average 8 years of age) (Fleisch et al., 2016). In a Massachusetts birth cohort with similar demographic characteristics to our study, Mao et al. (2016) found an increased risk of overweight (BMI z-score >= 85th percentile) and obesity (BMI z-score >= 95th percentile) in ages 2-9 years in the highest vs. lowest quartile of average prenatal $PM_{2.5}$ exposure (OR=1.3 (95% CI: 1.1, 1.6)) and postnatal $PM_{2.5}$ in the first two years of life (OR=1.2 (95% CI:1.1, 1.5)) (Mao et al., 2016). Jerrett et al. (2014) assessed associations between traffic density within 150 meters of the home longitudinal sex-specific BMI growth trajectories between the ages of 5 and 11 years of age, finding no significant association (Jerrett et al., 2014).

Discrepancies between our study and other studies that have assessed prenatal and postnatal $PM_{2.5}$ and postnatal distance to road measures with weight trajectories are complicated by differences in the temporality, type of exposure and outcome measures, and study population sociodemographic characteristics. For instance, we used 1 km^2 $PM_{2.5}$ predictions, while Chiu et al. 2017 used measures from the nearest monitor, which may have decreased exposure variability in their population. Other studies used BMI z-score and physiological measures of adiposity as their outcomes (Chiu et al., 2017; Fleisch et al., 2015; Mao et al., 2016; McConnell et al., 2015), whereas we used raw weight as our outcome measure. In the present study, we used raw-weights rather than z-scores to examine the true shape of the growth trajectory. Though McConnell et al.

(2015) and Jerrett et al. (2014) found positive associations between postnatal near roadway pollution and traffic density with rates of BMI growth, their exposure metrics incorporated traffic density and meteorological conditions, averaged over the year of each measurements whereas our study only looked at distance to road. Further, there was minimal distance to road variability in our study compared to that of McConnell et al. (2015) and Jerrett et al. (2014). Discrepancies between our results and their results may also be explained by lifestyle behaviors that affect traffic pollution exposure later in childhood, when these studies took place, while any relationship between traffic pollution and postnatal weight trajectories in early childhood in our study are likely a function of metabolic differences (McConnell et al., 2015).

Further, the CHW study population was more ethnically diverse and the prevalence of multiple hardships was higher as compared to the more ethnically homogenous and high-income VIVA cohort referenced in Fleisch et al. (2015, 2016). Inconsistencies may also be explained by our weaker measure of smoking (ascertained at the time of survey by asking whether caregiver smoked in the last 5 years), which has consistently been associated with weight, and has been shown to have a synergistic effect with air pollution in increasing growth rates (McConnell et al., 2015).

There are a number of limitations in this study. We did not have measures of maternal smoking during pregnancy and maternal pre-pregnancy BMI, both of which are risk factors for childhood weight gain (Linabery et al., 2013; Suzuki et al., 2015). We attempted to control for these measures using a variable measuring smoking status during the past five years and maternal BMI at study enrollment. CHW does not collect information on diet, physical activity, and other environmental exposures that may be jointly associated with air pollution and weight gain, so there may be some residual confounding in our analysis (Jerrett et al., 2010). To address this concern, we controlled for both a measure of hardship, including food insecurity, and block group median income, both of which were associated with child growth in our models. The distance to roadway measurement was not temporally resolved, which prevented us from taking into account multiple moves during the study period and may have biased our results towards the null.

There are some limitations inherent in the use of an EMR for data ascertainment. We were limited to residential addresses extracted from the EMR for exposure assignment starting at delivery, restricting our ability to account for moves during pregnancy and thus sensitive exposure periods. Weight measurements may be recorded differently by different health providers, resulting in non-differential outcome misclassification. However, weight measurements recorded in EMRs were found to be prone to <0.7% error from 0-5 years of age according to a large prospective cohort study using EMRs (Smith et al., 2010). Lastly, many of the visits in early childhood are routine checkups, whereas visits later in childhood may be comprised of children with poorer health outcomes, and thus differential susceptibility to the effects of $PM_{2.5}$ exposure later in life.

In spite of these limitations, our study has several strengths. This study included a large sample size: 4,797 participants, with 70,649 weight measurements. The analytical method used to model the appropriate function of weight for age allowed us to explore longitudinal differences in trajectories by levels of ambient air pollution exposure. This method further allowed us take full advantage of the EMR containing measurements collected at varying time points and frequencies, and to estimate change in slope during specified growth time periods. Using an EMR for epidemiological analyses is a novel, and relatively inexpensive source of longitudinal data ascertainment, allowing for a large number of measurements and limited potential for recall and participation bias (Casey et al., 2016). The CHW cohort is ethnically diverse and extremely mobile, making our results more representative of vulnerable populations that understudied in this body of literature. Survey data collected in the CHW also provided rich covariate information on multiple hardships, immigration status and caregiver characteristics. The validated exposure estimates used in our study are temporally resolved, allowing postnatal rolling average estimates to take into account multiple residential moves. Up-to-date parcel-level reference data used in the geocoding process also strengthened confidence in exposure assessment.

Our findings, and those of other studies examining early childhood weight trajectories, have multiple important public health implications. The associations in females are consistent with other risk factors implicated in the "thrifty phenotype" of low birthweight followed by rapid weight gain observed among females in this study (Stettler et al., 2002). This phenotype has been linked to several morbities in adulthood, including obesity, metabolic syndrome, type 2 diabetes and cardiovascular disease (Vaag et al., 2012). Although the differences in weight between $PM_{2.5}$ levels were small, the ubiquity of air pollution exposure across the population implies that even low levels of $PM_{2.5}$ may shift population prevalence of obesity over the life course. Differential weight trajectories, as noted here, have been shown to track into late childhood and adulthood (Giles et al., 2015). Our results demonstrate a period in which intervention could alter susceptibility to the effects of $PM_{2.5}$ through interventions known to promote healthy growth during the early childhood period, such as breastfeeding and introduction of a healthy diet (Braun et al., 2016).

Conclusion

We found in our study that low-birth weight females are at increased susceptibility for weight gain in early childhood when exposed to higher prenatal $PM_{2.5}$, with a significant inverse association among males. Studying growth trajectories, rather than attained measures of birth weight and BMI, provide an opportunity to understand susceptible phenotypes and periods of potential interventions. We found that air exchange rate, a measure of home leakiness, modified the effects of prenatal $PM_{2.5}$ exposure on growth in females. Therefore, mitigating the effects of prenatal $PM_{2.5}$ and traffic pollution exposure during pregnancy could include tightening homes with simultaneous controlled ventilation in the absence of significant indoor pollution sources (Fabian et al., 2012b; Macneill et al., 2014; Zota et al., 2005). Community interventions are also an important part of mitigating air pollution exposure during pregnancy and early childhood, especially among low-income communities. Such interventions may include increased use of land-use buffers, increasing vegetation and park locations, increasing active-travel locations such as bike and walking paths, and putting decking over highways, which are mutually beneficial to both decrease air pollution exposure and decreasing pre-pregnancy and pregnancy BMI by promoting exercise (Brugge et al., 2015). Because of the unique risk patterns found in the CHW population, further studies are needed in a variety of different study populations and geographies to replicate our findings. Future studies should also consider extending the follow-up period through adulthood and the implications of specific growth trajectory phenotypes on adult morbidities, such as cardiovascular disease and diabetes.

Supplement

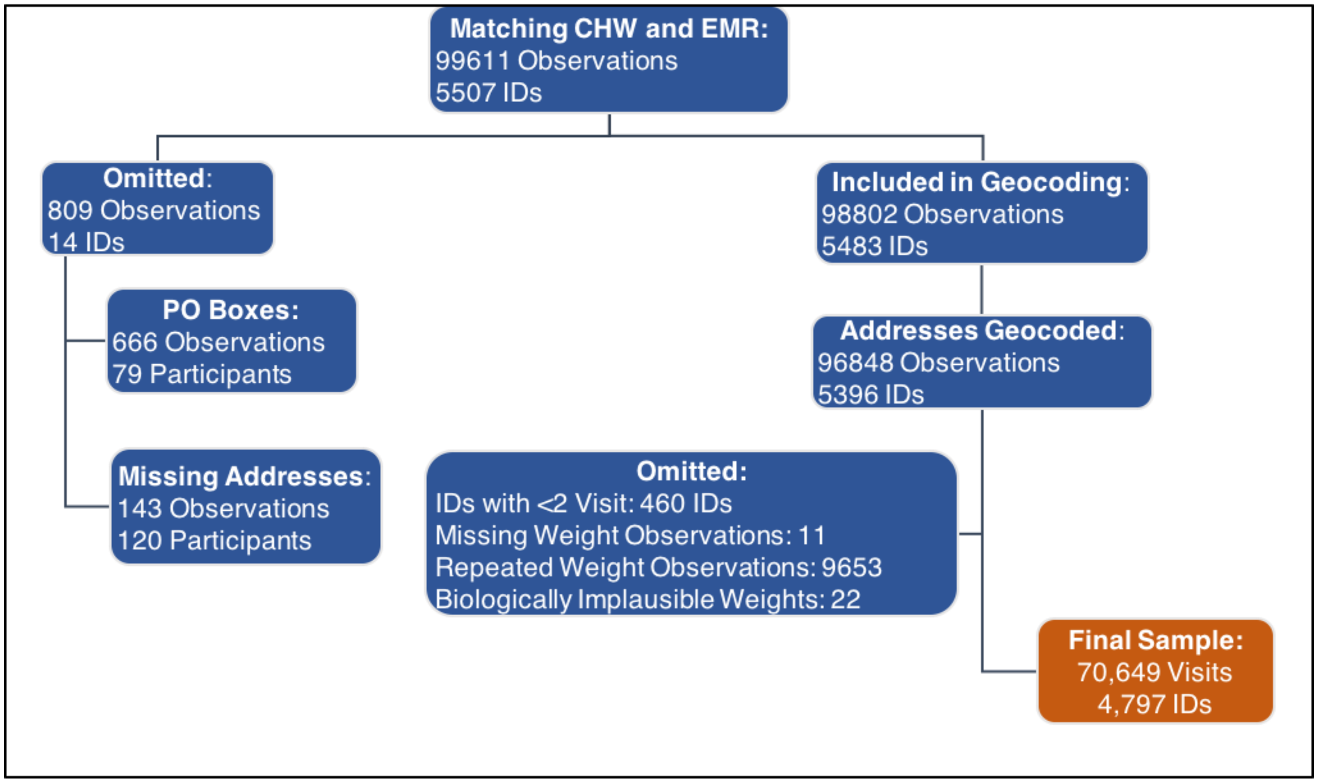


Figure S4.1. Analytical sample selection from linked Children's HealthWatch survey and electronic medical record data
Abbreviations: CHW (Children's HealthWatch); EMR (Electronic Medical Record)

Males[a] Females[a]

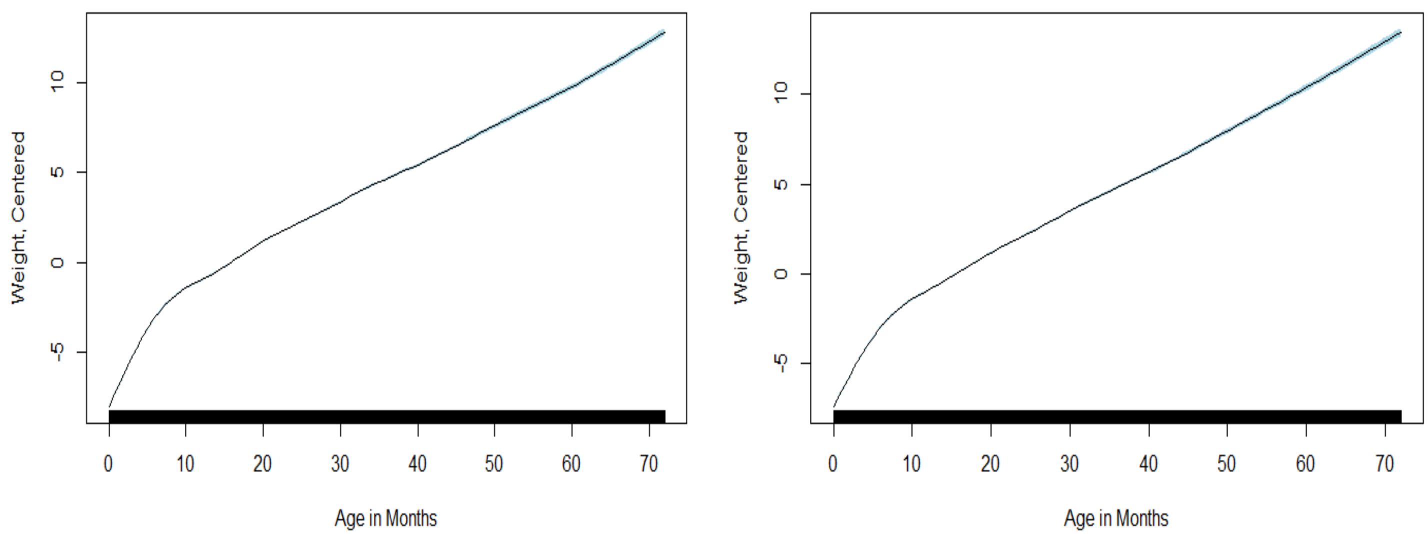


Figure S4.2. Cubic regression splines generated from generalized additive models adjusted for gestational age, cumulative hardship, median block group income, and ethnicity with 95% confidence intervals illustrating weight (kg) growth by age

[a]y-axis is centered at the mean; 95% confidence intervals (in light blue) are very small and are only visible towards the end of the growth curve

Table S4.1. Average Weight Growth Rate at Different Ages, by Sex, (kg/month)				
	Males (n=2213)		Females (n=1866)	
	Est.	95% CI	Est.	95% CI
0-3 months	0.99	(0.87, 1.11)	0.66	(0.52, 0.80)
3-6 months	0.61	(0.36, 0.85)	0.66	(0.37, 0.95)
6-12 months	0.32	(0.11, 0.54)	0.31	(0.06, 0.56)
12-24 months, males 12-18 months, females	0.18	(0.07, 0.29)	0.13	(-0.04, 0.30)
24-72 months, males 18-72 months, females	0.26	(0.21, 0.31)	0.21	(0.12, 0.31)

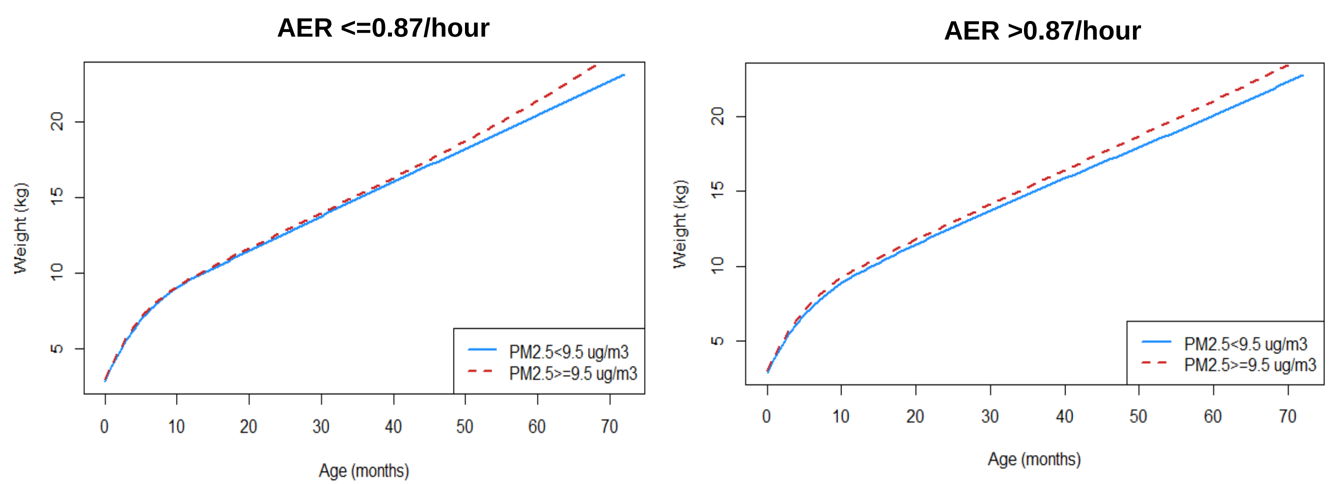


Figure S4.3. Predicted weight (kg) over age (months) by levels of average prenatal $PM_{2.5}$, stratified by air exchange rate (AER) categories
Models adjusted for: age, age^2, age^3, quadratic spline terms at 6 and 12 months, gestational age, ethnicity, education, immigrant status, cumulative hardship and block group median income

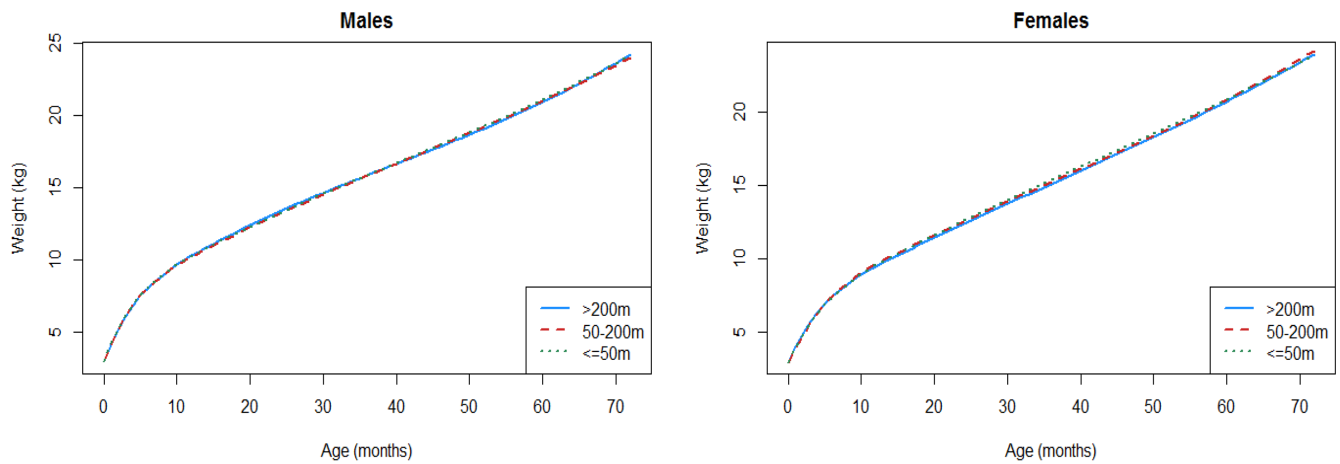


Figure S4.4. Predicted weight (kg) over age (months) by distance to major road
Models adjusted for: age, age^2, age^3, quadratic spline terms at 6 and 12 months, gestational age, ethnicity, education, immigrant status, cumulative hardship and block group median income

CHAPTER 5. CONCLUSIONS

The research summarized in this dissertation utilizes novel approaches to studying ambient air pollution inequalities in Massachusetts and to studying associations with ambient exposures and weight outcomes in early childhood. The work of *Chapter 2* revealed relative inequities in benefits gained by air pollution reduction strategies, especially in urban areas. *Chapter 3* demonstrated that residential characteristics are important modifiers of exposure to $PM_{2.5}$ of ambient origin across the Massachusetts population, and that residential characteristics increased exposure variability and magnified observed exposure inequalities. In *Chapter 4,* we examined associations between prenatal and postnatal ambient air pollution and distance to major roads and weight growth trajectories in an urban, ethnically diverse population. We found significant associations between average prenatal $PM_{2.5}$ and weight growth trajectories, finding opposite directions of effect for females and males. Taken together, this work suggests that non-white and low-income communities continue to be disproportionately exposed to ambient air pollution, resulting in health consequences for vulnerable, urban populations.

Chapter 2. Temporal trends in air pollution exposure inequality in Massachusetts

The aim of *Chapter 2* was to quantify longitudinal changes of ambient $PM_{2.5}$ and NO_2 exposure inequalities across Massachusetts between 2000 and 2010. Using Census and ACS data, we first calculated urban, rural, and statewide population-weighted $PM_{2.5}$ and NO_2 concentrations. We found that ambient concentrations were highest for urban non-Hispanic black (11.8 $\mu g/m^3$ in 2003 and 8.4 $\mu g/m^3$ in 2010) and urban Hispanic (15.9

ppb in 2005 and 13.0 ppb in 2010) populations, for annual-average $PM_{2.5}$ and NO_2 respectively, compared to all other population subgroups. Absolute $PM_{2.5}$ and NO_2 decline was similar across population subgroups, but relative reductions of $PM_{2.5}$ and NO_2 disproportionately favored non-Hispanic Asian populations and households with a median annual income above $75,000 in rural areas.

We used a novel application of the Atkinson Index to assess longitudinal environmental inequalities. We found that $PM_{2.5}$ and NO_2 inequality was higher among urban and statewide racial/ethnic groups compared to income and education groups. $PM_{2.5}$ inequality fluctuated over the study period, but was higher overall in urban compared to rural communities, and NO_2 inequality increased over the study period, despite overall concentration reductions. We found that concentration distributions, and not population mobility, likely explains observed longitudinal inequality trends in urban settings.

These results corroborate a recent nationwide study of longitudinal NO_2 inequalities, stratified by rural and urban block groups (Clark et al., 2017). The authors found that NO_2 inequalities were higher in magnitude for racial/ethnic than among other demographic characteristics, and that relative NO_2 inequalities persisted over the study period. The authors also found that changes in NO_2 distributions contributed more heavily than demographic patterns to observed inequalities over the study period.

This study improves upon existing environmental inequality literature by assessing longitudinal environmental inequality trends at a relatively small spatial scale for two separate pollutants. This study also adds to the environmental inequality literature by using temporally and spatially resolved $PM_{2.5}$ and NO_2 estimates, which allowed us to separately examine population and air pollution patterns to determine which best explains increasing inequality.

Chapter 3. The impact of AER on ambient air pollution exposure and inequalities across all residential parcels in Massachusetts

The aim of *Chapter 3* was to develop a measure of home-leakiness that could be applied as a modifier in studies that utilize ambient concentrations as exposure proxies. A principal limitation in many studies of air pollution exposure is lack of information on residential characteristics that influence exposure variability. Certain residential characteristics, such as home leakiness, may exhibit sociodemographic gradients.

We estimated AERs for all residential parcels by using the LBL model, which estimates AER as a function of house age, area, height, surrounding meteorological conditions and density of surrounding obstructions (Breen et al., 2014, 2010; Sarnat et al., 2013). We expanded the LBL model, which was originally developed for single-family homes, to estimate AER for units in multi-family buildings by assigning a multi-family parcels a shelter class of "5". This approach accounts for maximum surrounding obstructions as a proxy for fewer externally-facing walls. This model was estimated using easily-accessible publicly available data. We found that indoor concentrations of ambient origin exhibited variability across residential parcels, which was twice that of outdoor ambient concentrations. The flexibility of the LBL model allows for extrapolation to a range of other outdoor-generated pollutants provided information about decay rate and penetration efficiency.

We performed an inequality analysis quantifying demographic characteristics of block groups containing parcels in the 10th (low-exposed) and 90th (high exposed) percentiles of the individual and combined $PM_{2.5}$ and AER exposure distributions. Parcels in the 90th percentile of $PM_{2.5}$ were 75% non-Hispanic white, and 11% Hispanic, compared to parcels in the 10th percentile of $PM_{2.5}$, which were 94% non-Hispanic white and 2% Hispanic. Similar results were found for AER distributions: block groups containing parcels in the 90th percentile were 14% Hispanic and 68% non-Hispanic white, compared to 3% Hispanic and 90% non-Hispanic white for those in the 10th percentile.

Inequality was magnified for distributions of parcels in the 10th and 90th percentiles of combined $PM_{2.5}$ and AER. These results reveal a disproportionate burden of leakier homes in areas of higher $PM_{2.5}$ concentrations for non-white and low-income populations. For instance, block groups containing "high exposed" parcels were, on average, 22% Hispanic, versus 10% of the state population. Similar results were found for non-Hispanic black populations and among low-income groups.

In summary, these findings suggest that using ambient air pollution as an exposure surrogate in the absence of inter-individual housing variability may underestimate associations found in both epidemiology and inequality studies. We also found that that non-Hispanic white and high-income populations live in homes with lower AER, the distribution of which has not previously been characterized.

Chapter 4. Prenatal and postnatal ambient air pollution: association with longitudinal weight growth trajectories in early childhood

The aim of *Chapter 4* was to examine associations between prenatal and postnatal $PM_{2.5}$ and distance to major roadway and early childhood weight growth trajectories. We employed novel statistical approaches to approximate weight-for-age trajectories in the study population and examine associations with exposures of interest. These statistical approaches allowed us to visualize divergent weight trajectories by levels of prenatal $PM_{2.5}$ and distance to road, and to assess the postnatal relationship as change in growth rates (weight for age) for every 10 $\mu g/m^3$ increase postnatal $PM_{2.5}$.

Males had significantly lower weights in the high prenatal $PM_{2.5}$ group compared to weights in the low prenatal exposure group after 24 months. We find the opposite effect in females, where participants with $PM_{2.5}$ above the median had significantly higher weights than the low $PM_{2.5}$ group throughout the full growth trajectory.

Stratified analyses by birth weight revealed that the difference in growth trajectories strengthened among LBW females, but was attenuated in non-LBW females, indicating increased susceptibility to the effects of $PM_{2.5}$ among the LBW female group. Results did not differ between male birth strata. When stratified by AER in the subset for which AER values were estimated, the absolute different in weight between $PM_{2.5}$ levels in the high AER group was approximately twice that of the low AER group in females between 6 and 48 months. In both strata, weights were higher for participants with $PM_{2.5}$ above the median. The interaction of AER with prenatal $PM_{2.5}$ was not significant in boys. This finding is supported by previous literature demonstrating that females are more susceptible to obesity under increasing measures of hardship in childhood (Khlat et al., 2009).

Limitations

This section summarizes shared limitations in *Chapters 2-4.* Detailed discussion of study limitations can be found in individual chapters.

Utilizing gridded $PM_{2.5}$ and NO_2 predictions across all three chapters as proxies for true exposure ignores inter-individual variability in time-activity patterns. This approach may result in some exposure misclassification, especially for NO_2 which exhibits more local variability. This misclassification may ignore household, occupational and commuting microenvironments that are differential by racial/ethnic and income groups (O'Neill et al., 2003; Su et al., 2011). Though these models have demonstrated high predictive reliability when validated against ambient $PM_{2.5}$ and NO_2 measurements, future studies are needed comparing concentrations estimates with personal exposures.

As a common overall limitation to *Chapters 2-4,* we omit potential influences of indoor-generated pollutants as part of our exposure estimates, thus ignoring other important sources of variability. Considering the full exposure profile of both indoor and outdoor-generated air pollutants may reveal a more striking characterization of exposure inequality between population groups in *Chapter 2*. In *Chapter 3*, homes with high AER have higher indoor concentrations of outdoor-generated pollutants, but will have reduced influence from indoor sources, such as tobacco smoke and combustion. This same limitation applies to our findings in *Chapter 4* of larger weight differences among females living in homes with higher AER. In this chapter, high AER may be indicative of poor housing quality, which has independently been associated with poor metabolic outcomes (Hood, 2005; Jacobs et al., 2009). However, we were still unable to control for contributions from indoor sources that may be differential by both exposure groups and levels of the outcome in our study population, with the exception of smoking status.

The spatial unit at which Census data was available also limited our analysis. We assigned uniform concentrations to all individuals and households within the same block group to approximate individual exposure in *Chapter 2 and 3*. However, unlike unit-hazard coincidence methods, these exposure assignments are not limited by geographic unit boundaries because the $PM_{2.5}$ and NO_2 predictions take into account meteorological conditions and chemical fate and transport.

Using publicly available data in *Chapters 2 and 3* inherently limits the temporality at which the data are available. In *Chapter 2* there was temporal misalignment between predicted $PM_{2.5}$ and NO_2 concentrations and available Census data. In *Chapter 3* there was temporal misalignment between the parcel data, which was updated in 2015, 2010 meteorologic data, and land-use polygons which were updated in 2005. The stability of our inequality measures in *Chapter 2* across Census data indicates that this is a minor source of error. We are still confident in our estimates of AERs calculated for the year 2010, as parcel and land-use classifications are not expected to rapidly shift over time.

Public Health and Policy Implications

Despite improved technological advances, financial incentives and community-level interventions aimed at decreasing ambient air pollution concentrations, ambient air pollution still remains a significant public health burden. Several morbidities that have been linked to ambient air pollution exposure are becoming increasingly prevalent in the U.S. (Akinbami et al., 2012; Eze et al., 2015; Gregg and Shaw, 2017). Respiratory outcomes, cardiovascular disease and premature mortality have been associated with ambient air pollution at levels below the current NAAQS (Calderón-garcidueñas et al., 2015; Eze et al., 2015; Shi et al., 2016; Smith and Peel, 2010). Concentrations in *Chapter 4* were, on average, lower than the NAAQS. The existence of significant associations in our studies and many others conducted in areas below the NAAQS suggests a need to re-examine the NAAQS to determine if it is adequately protective of sensitive subpopulations, especially given novel outcomes and subpopulations not previously central to this process.

In addition to strengthening current standards, community-level interventions should be targeted at communities that disproportionately face cumulative health impacts of air pollution sources and social stressors (Morello-Frosch et al., 2011). For example, increased air quality monitoring in areas with poor air quality, decreased local industrial siting incentives and increased oversight over existing regulations in vulnerable communities may all reduce inequitable burdens (Miranda et al., 2011; Morello-Frosch, 2002). Air pollution reduction approaches should be jointly implemented with health-promoting resources to reduce health disparities. The results of *Chapter 4* demonstrate potential windows in early development at which these health promotion and pollution reduction approaches may be most effective in decreasing obesity development. Incorporating quantitative measures of inequality, as demonstrated in *Chapter 2,* are also powerful tools for policymakers to understand spatial distribution of inequities and how different control scenarios could decrease exposure in vulnerable communities (Harper et al., 2013; Levy et al., 2007).

The home environment is one factor of great importance in mitigating exposures in vulnerable populations. Segregated non-white and low-income communities tend to be concentrated in areas with poorer housing stock, higher proportions of renters, and fewer resources to improve housing stock, making interventions at the housing level to reduce environmental exposure particularly challenging (Crocker et al., 2011; Morello-Frosch and Shenassa, 2006; Rosofsky et al., 2016). We found here that non-white and low-income populations are more likely to live in leakier homes in areas with high pollution.

Interventions that improve housing stock to decrease infiltration of ambient pollutants indoors may decrease observed exposure inequalities and eventual health disparities. Such interventions must comprehensively address indoor and outdoor pollutant sources (Adamkiewicz et al., 2011; Morello-Frosch et al., 2011). For instance, homes sealed to decrease infiltration of outdoor contaminants should be concurrently outfitted with improved ventilation to reduce exposure to indoor contaminants.

Directions for Future Work

In *Chapter 2* we demonstrated a need for exposure inequality studies that examine longitudinal trends between different sub-populations. More research in other geographic areas with different ambient concentrations is needed to corroborate our findings in Massachusetts and those of Clark et al. (2017), which took place nationwide.

Large sample sizes are required for environmental inequality studies to elucidate the driving mechanisms. Consequently, most exposure inequality research is limited by the spatial scale and data collected by the U.S. Census and other publicly-available population-level datasets. There is potential in making greater use of longitudinal cohort studies for exposure inequality research, as has been demonstrated in a limited number of studies (Crowder and Downey, 2010; Downey et al., 2017; Mohai et al., 2009; Pais et al., 2014). Cohort studies provide access individual-level covariate data and accurate exposure classification with geocoded addresses to provide detailed insight into the interplay between individual-level behavioral and evolving neighborhood factors that influence exposures (Mohai and Saha, 2015a; Morello-Frosch and Jesdale, 2006).

Community-based participatory research (CBPR) has great value in exposure inequality research. CBPR actively engages community members and organizations to develop research questions and collect data. Community members can provide key perspectives about longitudinal mechanisms of exposure inequality and perceptions of the changing physical and social environment. CBPR can be especially effective for studies of weight gain and obesity to understand how local residents interact with the physical environment, environmental exposures and residential-based obesity risk factors (Israel et al., 2005; Lopez and Hynes, 2006). Consultations with local residents can bring questions to light that may not have been immediately apparent to investigators, and simultaneously empower communities to influence local policy to reduce inequities.

Our research on early-life ambient air pollution exposure and weight growth in childhood is one of a new and growing field on patterns and risk factors of weight growth trajectories (Howe et al., 2015). Further studies should be undertaken in the environmental health field that examines longitudinal weight trajectories, rather than cross-sectional outcomes to establish whether early-life exposures that impede fetal growth persist over the life course. Specific early-childhood growth trajectory phenotypes, such as the “thrifty” phenotype, have been linked to adverse cardiometabolic outcomes in adulthood (Hales and Barker, 2001; Vaag et al., 2012). Our study found that $PM_{2.5}$ was associated with different trajectories in males and females, with faster growth rates and higher weights overall in the high-$PM_{2.5}$ exposed female group. Future research identifying growth trajectory phenotypes or rates of growth that are more predictive of these chronic outcomes can inform whether observed weight growth trajectories are harmful or beneficial to long-term health (Barker et al., 2005), and relatedly, the public health implications of either increased or decreased growth trajectories among various subpopulations.

Future work in the CHW cohort will extend these findings by exploring other potential modifiers of the association between air pollution exposure and growth trajectories, for which a robust set of variables is available. These include measures of greenness, residential segregation, immigration status, breastfeeding practices, homelessness, and other measures of hardship, all of which have been understudied in this body of literature.

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