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Health disparities attributable to air pollutant exposure in North Carolina: Influence of residential environmental and social factors

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ABSTRACT

Understanding the environmental justice implications of the mortality impacts of air pollution exposure is a public health priority, as some subpopulations may face a disproportionate health burden. We examined which residential environmental and social factors may affect disparities in the air pollution-mortality relationship in North Carolina, US, using a time-stratified case-crossover design. Results indicate that air pollution poses a higher mortality risk for some persons (e.g., elderly) than others. Our findings have implications for environmental justice regarding protection of those who suffer the most from exposure to air pollution and policies to protect their health.

1. Introduction

A large body of literature has demonstrated consistent evidence of the effects of exposure to air pollution on mortality (Achilleos et al., 2019; Di et al., 2017; Qu et al., 2018; Wu et al., 2019; Yu et al., 2019). Such mortality burdens may vary by population and region. However, questions remain on which individual and community factors contribute to differences in the associations between air pollution and health among subpopulations. Understanding these health disparities and their potential determinants is a critical public health concern.

Recent studies on disparities suggest that several factors such as sex, age, pre-existing conditions, race/ethnicity, socioeconomic status (SES), and residential environmental factors such as proximity to green spaces and blue spaces may be associated with higher risk of adverse health outcomes related to exposure to air pollution (Li et al., 2017; Liu et al., 2019; Ou et al., 2008; Qu et al., 2018; Tibuakuu et al., 2018). For example, a study in Hong Kong found that female, the elderly, and people with lower SES had increased risk of death associated with air pollution compared to other populations (Qiu et al., 2015). Another study by Richardson et al. (2013) found that persons in lower-income regions in Europe were more susceptible to the health effects of PM_{10} than other populations, however the findings varied between Eastern and Western Europe, and by type of mortality. Place or neighborhood factors may play an important role in explaining spatial heterogeneity in air pollution exposure and/or health risk. Living in different residential areas may lead to differential exposure to stressors and access to neighborhood resources (Gee and Payne-Sturges, 2004). Although some studies suggested health disparities from the impacts of exposure to air pollution, further work at different locations is needed given the variation in population characteristics across regions and the potential changes in disparities over time given temporal patterns in related variables (e.g., air pollution levels, population structure). Identifying the most important factors related to disparities to air pollution-mortality associations in a given location and the most affected subpopulations is critical to establish appropriate plans and conduct effective interventions to protect public health.

Ozone and $PM_{2.5}$ are major atmospheric pollutants directly affecting human health. The recent Global Burden of Disease (GBD) estimated that exposure to ambient $PM_{2.5}$ causes 4.2 million deaths globally, with an additional 254,000 deaths globally caused by ozone exposure (GBD, 2015). US burden of disease study lists ambient $PM_{2.5}$ and O_3 pollution as the 8th and 15th leading risk factors in the US in 2010 (Murray and Collaborators, 2013). Numerous studies in many parts of the world provided scientific evidence that increased risk of mortality was associated with exposure to these pollutants (Fann et al., 2012; Farhat et al., 2013). The aim of our study was to assess several health disparity factors for major air pollutants with mortality associations. Thus, we chose $PM_{2.5}$ and O_3 as our key exposure of interest.

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Recent studies have used predicted air pollutant concentrations, which allow for better spatial and temporal coverage than monitoring data, to estimate the relationship between ambient air pollution and several health outcomes (Bravo et al., 2017; Fann et al., 2018). While these values are estimates, they address the lack of high spatial and temporal resolution in many ambient monitoring networks. Most monitors are located in urban areas, which may not fully reflect exposure in rural regions without monitors. Also, many monitors do not operate continuously throughout the year (e.g., measurements every 3 or 6 days for $PM_{2.5}$, only for the warm season for O_3), which prohibits the investigation of cumulative acute exposures over multiple days. Limited spatial and temporal resolution of some monitoring networks may hinder investigation of exposure and health effects in some regions. Thus, health effect estimates based on monitoring data alone may not fully capture the susceptibility due to differences between communities or subpopulations.

North Carolina (NC) is relatively large and diverse state with underlying geographies that include extensive agricultural regions and forests, coastal areas, and multiple medium-large urban centers. NC has a range of air quality with areas in noncompliance with EPA regulations for criteria pollutants (e.g., O_3, $PM_{2.5}$) and distinct spatial patterns of racial distribution or poverty patterns. This study area allows us to evaluate diverse populations and factors regarding environmental health disparities. Although previous studies in this area explored the relationship between air pollution, race, and SES (Gray et al., 2013) or investigated spatial-temporal association between $PM_{2.5}$ and daily mortality (Choi et al., 2009), no study evaluated several residential environmental and socioeconomic factors that may affect disparities in air pollution-mortality relationships and assessed multiple disparities, which can contribute to a better understanding of interactions of disparity factors.

This study investigated the health disparities attributable to exposure to air pollutants ($PM_{2.5}$, O_3) in North Carolina, USA. We used Community Multi-scale Air Quality (CMAQ) downscaler output to estimate daily $PM_{2.5}$ and O_3 concentrations for 2002–2013. We evaluated which residential environmental and socioeconomic factors affect disparities in air pollution-mortality relationships using a stratified model for each effect modifier. Our study has implications for environmental justice regarding which subpopulations are vulnerable and which factors affect disparities in associations between air pollution and mortality. This work extends current understanding of environmental health disparities.

2. Methods

2.1. Data

We obtained individual-level mortality data for North Carolina from 2002 to 2013 from the North Carolina State Center for Health Statistics, Vital Statistics Department. For each participant, mortality data included date of death, residential location, sex, age at death (<65, ≥ 65 years), race/ethnicity (non-Hispanic white, non-Hispanic black, Hispanic, non-Hispanic Asian, or non-Hispanic other), education (<12 years, high school graduate, 1–4 years of college, ≥ 5 years of college, or unknown), and marital status (never married, married, widowed, divorced, or unknown). We excluded participants with incomplete data for any variable. We classified mortality data as: total mortality as all causes of death except external causes (International Classification of Diseases, ICD-10, A00-R99), cardiovascular mortality (ICD-10, I00-R99), and respiratory mortality (ICD-10, J00-J99).

Ambient $PM_{2.5}$ and O_3 concentrations for each of North Carolina's census tracts were obtained for 2002–2013 from the downscaler output from the US Environmental Protection Agency (EPA). The downscaler utilizes air monitoring station data and Community Multiscale Air Quality (CMAQ) output at 12 × 12km grid cell resolution to estimate daily air pollution concentrations at census tract centroids. Downscaler output includes estimates of daily 24-h average for $PM_{2.5}$ and daily 8-h maximum for O_3. For these estimates we assigned exposure based on the grid cell in which the participant's residence was located. Additional details for the downscaler modeling approach and evaluation are provided elsewhere (Berrocal et al., 2012). To compare the robustness of effect estimates of downscaler predicted $PM_{2.5}$ and O_3 levels with those generated using monitoring data, we obtained daily 24-h $PM_{2.5}$ and 8-h maximum O_3 measurement values from the EPA's Air Quality System. We assigned exposures for each participant as the daily measurements from monitors nearest each subject's residence (based on each subject's residential location (latitude/longitude)) including monitors outside North Carolina, within 40 km of North Carolina's boundary. We used the downscaler exposure estimates as main analysis and monitor-based values as sensitivity analysis. The total number of cases (i.e., deaths) for downscaler- and monitor-based estimate was 775,338 and 209,669, respectively. There were fewer deaths when using monitor-based estimates as monitor-derived air pollution effect estimates were based on the time and locations for which exposure estimates are available from both methods.

Due to lack of measured daily weather data, we used gridded weather data at the county level. The gridded weather data using Parameter-elevation Regressions on Independent Slopes Model (PRISM) interpolation method are reported on a daily basis and at high spatial resolutions (4 × 4km grid). PRISM provides data for the continental US. The algorithms and the details have been described elsewhere (Daly et al., 2008; PRISM Climate Group, 2015). A previous study showed good agreement between measured and gridded weather data (Mourtzinis et al., 2017). We used daily levels of temperature and dew point temperature at the county level. County-level values were calculated as the average of all grid cells with centroids within each county.

To assess health disparity factors in the association between exposure to air pollution and mortality, we included several residential environmental and socioeconomic factors based on the previous literature review. We considered individual-level factors, residential greenness, proximity to water bodies, median household income, and classification of urbanicity.

As a residential greenness measure, urban vegetation was assessed using the Normalized Difference Vegetation Index (NDVI) derived from the Moderate Resolution Imaging Spectroradiometer (MODIS) sensor aboard the Terra satellite image from NASA's Earth Observing System. We used the global MODIS product MOD13Q1 version 5, which has been corrected for atmospheric contamination from water, clouds, and aerosols. This product is a 16-day composite at a spatial resolution of 250 m. We calculated average NDVI at the ZIP-code level for the study period. We categorized average NDVI as above or below the median (<0.61 or ≥ 0.61).

We obtained information on water bodies (e.g. river, lake) from the North Carolina Department of Environmental Quality Online GIS to examine effect modification by proximity to water bodies. We calculated the distance from each subject's residence to water bodies to assess the effect of blue space and categorized proximity to water as above or below the median (<10.2 km or ≥ 10.2 km).

To assess community-level effect modification we used 2010 Census data at the census-tract level including variables of median household income, as a surrogate for SES, and population size. We classified urbanicity as metropolitan (urban area $\geq 50{,}000$ people), micropolitan (urban cluster of 10,000–49,999), and rural (urban cluster of $<10{,}000$) area. We used median values to define categories of income.

2.2. Statistical analysis

We applied a time-stratified case-crossover design to estimate the association between air pollution and mortality. In this approach, each case acts as his or her own control and thus the method has benefits of controlling for potential confounding from fixed characteristics by design. To avoid selection bias, we applied time-stratified referent

selection based on same day of the week of the same year when a death occurred. Each case could be compared to multiple control days.

Some O_3 monitors operate only during the warm season (e.g., April–September) when O_3 is anticipated to be high. We generated separate effect estimates for the association with mortality for: 1) year-round O_3 exposure and 2) warm season O_3 exposure (April–September). We examined the lagged effect of air pollutants with single-day lags (lag 0, lag 1, lag 2) and multi-day lags (lag 01, lag 02). Lag 0 meant the effect of the air pollution on the same day as the day of mortality (i.e., date of death). Lag 1 refers to the air pollution on the day before the day of death. Lag 02 presented the cumulative effect of the current day and prior 2 days' air pollution on the current day's mortality. For monitor-based effect estimates, we investigated the effect of $PM_{2.5}$ for only single-day lag as most $PM_{2.5}$ monitors typically record observations every three days.

We conducted additional analyses considering spatial clustering in the model. We accounted for spatial autocorrelation from unmeasured spatially distributed risk factors by including a random intercept for each county where cluster effects are incorporated into the model as independent and identically distributed random variables to account for the within-cluster correlation. We also conducted additional analysis considering NDVI at the county level.

We calculated Population-attributable risks (PARs) based on the calculated effect estimates in this study. The PAR% is the percentage of incidence of a disease within a population (exposed and non-exposed), due to exposure. This statistic describes the percentage incidence of a disease within a population that could be prevented if exposures were eliminated. We estimated PARs per pollutant using our risk estimates and the following equation: $PAR\% = 100 \times P(R-1)/[P(R-1)+1]$, for which P is the prevalence of the exposure (i.e., air pollution) in the population and is assumed to be 100% as everyone in the population exposed to air pollution and R is the relative risk (or OR).

To examine the potential effect modifiers, we performed stratified analyses by individual- and community-level factors for total mortality. We then tested statistical significance of differences between effect estimates of strata of a potential effect modifier by calculating the 95% confidence interval as $(Q_1\text{-}Q_2)\pm 1.96\sqrt{SE_1{}^2 + SE_2{}^2}$ where Q_1 and Q_2 are the estimates for the two strata of the potential effect modifier (e.g., male and female), and SE_1 and SE_2 are their respective standard errors. To categorize community-level factors, we tested other cutoff points (e.g., quartile) as well as median value. We also investigated multiple susceptibilities by combinations of potential factors of effect modification (e.g., race/ethnicity and census-tract median income). We fitted conditional logistic regression models to estimate the association between air pollution and mortality. Odds ratios and 95% confidence intervals were calculated on the basis of an increase of 10 $\mu g/m^3$ in $PM_{2.5}$ or 10 ppb in O_3. All analyses were conducted using SAS (9.4, SAS Institute, Cary, NC, USA) and R (version 3.5.1, R Core Team).

3. Results

During the study period, there were 775,338 cases (i.e., total deaths) with 3,410,015 control days. Table 1 shows characteristics of the study population. The 775,338 total deaths included 261,663 from cardiovascular disease and 86,017 from respiratory disease. The study population had more females than males (52.3% vs. 47.7%). The majority of the deceased were non-Hispanic white (77.7%), and ≥65 years (75.2%). Most subjects had less than a high school level education or were high school graduates (71.2%) and were married or widowed (78.5%). For community-level characteristics, mean census-tract median income was $45,116. Most subjects lived in metropolitan areas (85.0%). For study participants, average NDVI was 0.61 and average distance from residence to water bodies was 12.6 km.

The average $PM_{2.5}$ concentrations from CMAQ downscaler and EPA monitor were similar, although average O_3 concentration for CMAQ downscaler output was higher than EPA monitor concentrations. Spatial variations in air pollution levels are provided in Fig. 1. Descriptive statistics for residential environmental factors are provided in Supplementary Table 1 and air pollution levels based on community-level SES are provided in Supplementary Table 2.

Table 1
Characteristics of study population in NC, 2002–2013.

Characteristics	Statistics
Cause of death	
Total	775,338
Cardiovascular	261,663
Respiratory	86,017
Sex (N, %)	
Male	369,883 (47.7)
Female	405,441 (52.3)
Missing	14 (0.0)
Race/ethnicity (N, %)	
Non-Hispanic White	602,125 (77.7)
Non-Hispanic Black	158,449 (20.4)
Hispanic	5307 (0.7)
Non-Hispanic Asian	3239 (0.4)
Non-Hispanic Other	6096 (0.8)
Missing	122 (0.0)
Age at death (N, %)	
<65 years	192,631 (24.8)
≥65 years	582,707 (75.2)
Education (N, %)	
<12 years	303,198 (39.1)
High school graduate	249,042 (32.1)
1–4 years of college	175,497 (22.6)
5 or more years of college	36,141 (4.7)
Unknown	11,460 (1.5)
Marital status (N, %)	
Never married	73,592 (9.5)
Married	308,906 (39.8)
Widowed	299,738 (38.7)
Divorced	91,856 (11.9)
Unknown	1246 (0.2)
Community-level factors	
Census-tract median income ($, mean ± SD)	45,116±18,015
County-level urbanicity (N, %)	
Metropolitan (urban area ≥50,000 people)	659,332 (85.0)
Micropolitan (urban cluster of 10,000–49,999)	113,692 (14.7)
Rural (urban cluster of <10,000)	2314 (0.3)
Average NDVI (mean ± SD)	0.61 ± 0.05
Distance to water bodies (km, mean ± SD)	12.6 ± 10.3
Air Pollution Estimates (mean ± SD)	
Downscaler $PM_{2.5}$ ($\mu g/m^3$)	11.4 ± 5.7
Downscaler O_3 (ppb)	41.7 ± 13.7
Monitor-based $PM_{2.5}$ ($\mu g/m^3$)	11.5 ± 6.1
Monitor-based O_3 (ppb)	31.3 ± 11.1
Weather variables (mean ± SD)	
Temperature (°C)	14.9 ± 8.7
Dew point temperature (°C)	8.2 ± 9.8

Supplementary Table 3 provides correlation coefficients across variables. Strong positive correlations between downscaler- and monitor-derived air pollution concentrations were observed ($r = 0.96$ for $PM_{2.5}$; $r = 0.84$ for O_3). Average NDVI was negatively correlated with county-level total population ($r = -0.48$).

Table 2 shows odds ratios (OR) and 95% confidence intervals (CI) of the association between exposure using downscaler-derived $PM_{2.5}$ and O_3 for risk of total and cause-specific mortality. All models were adjusted for same day's temperature and dew point temperature. We observed positive associations between $PM_{2.5}$ exposure and risk of total and cardiovascular mortality. An 10 $\mu g/m^3$ increase in lag 01 $PM_{2.5}$ exposure was associated with an OR of 1.019 (95% CI 1.012, 1.025) and 1.017 (95% CI 1.007, 1.028) for total and cardiovascular mortality, respectively. For O_3, a 10 ppb increase in lag 02 exposure was associated with total mortality (OR 1.006; 95% CI 1.002, 1.010). We did not find statistically significant associations with respiratory mortality.

We performed additional analysis for the warm season (April to September) O_3 (Supplementary Table 4). The effect estimates from the

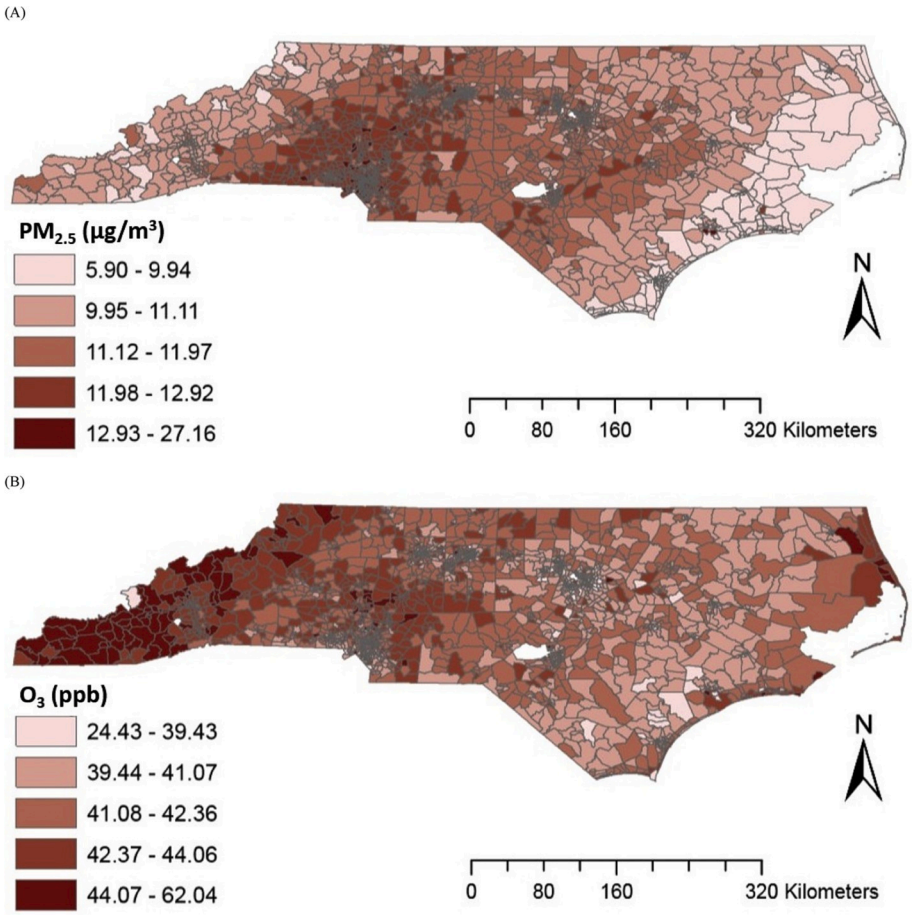

Fig. 1. Spatial variations in air pollution levels (A) $PM_{2.5}$ (B) O_3.

Table 2
Odds ratios and 95% confidence intervals of $PM_{2.5}$ and O_3 for total and cause-specific mortality.

	Total	Cardiovascular	Respiratory
Downscaler $PM_{2.5}$ (per 10 μg/m^3)			
Lag 0	1.015 (1.009, 1.020)	1.011 (1.002, 1.020)	1.004 (0.988, 1.020)
Lag 1	1.015 (1.009, 1.020)	1.016 (1.007, 1.026)	1.002 (0.986, 1.019)
Lag 2	1.004 (0.998, 1.009)	1.003 (0.993, 1.012)	0.998 (0.982, 1.015)
Lag 01	1.019 (1.012, 1.025)	1.017 (1.007, 1.028)	1.004 (0.986, 1.023)
Lag 02	1.018 (1.011, 1.025)	1.016 (1.004, 1.028)	1.003 (0.983, 1.025)
Downscaler O_3 (per 10ppb)			
Lag 0	1.004 (1.001, 1.006)	1.000 (0.995, 1.004)	1.003 (0.995, 1.012)
Lag 1	1.004 (1.001, 1.007)	1.003 (0.997, 1.008)	0.999 (0.989, 1.009)
Lag 2	1.003 (1.000, 1.006)	1.003 (0.998, 1.007)	1.000 (0.991, 1.008)
Lag 01	1.005 (1.002, 1.008)	1.001 (0.996, 1.007)	1.002 (0.991, 1.012)
Lag 02	1.006 (1.002, 1.010)	1.003 (0.996, 1.009)	1.001 (0.989, 1.012)

N for downscaler $PM_{2.5}$ and O_3 exposure: 775,338.

warm season were generally similar with those of year-round O_3.

To confirm the robustness of our findings, we performed additional analysis comparing the effect estimates based on exposures from the downscaler- and monitor-derived concentrations (Fig. 2). Effect estimates from the monitor-derived concentrations were similar to those of the original findings from downscaler-derived concentrations.

We observed positive associations between exposure to $PM_{2.5}$ and O_3 and risk of total mortality; for these associations we conducted additional analyses considering spatial autocorrelation in the model. Results were similar to original findings (Supplementary Table 5). We calculated PAR for air pollution and total mortality. The PARs for total mortality due to $PM_{2.5}$ (lag 01) and O_3 (lag 02) exposure were 1.9% and 0.6% respectively (Supplementary Table 6). Findings from additional analysis considering NDVI at the county level were similar with original findings (Supplementary Table 7).

For the exposure lag and mortality for which we observed the largest and also the most statistically significant associations (i.e., $PM_{2.5}$ and O_3 exposure for total mortality), we evaluated effect modification by community and individual characteristics. Table 3 shows estimated associations between air pollution exposure and total mortality stratified by community-level factors. We investigated the relationship between $PM_{2.5}$ and O_3 exposure and total mortality by residential green space, blue space, urbanicity, and census-tract median income level (Table 3). We did not find any statistically significant differences between groups. However, the association between air pollution and the risk of total mortality was slightly higher, although not statistically different, in areas with less green space, further distance to water bodies, ≥50,000 people, or lower median income level. Estimated associations for O_3 showed similar patterns except for urbanicity and census-tract median income level.

We also assessed potential effect modification by individual characteristics (Table 4). Stratified analyses showed that associations between $PM_{2.5}$ exposure and total mortality were higher in males than females although these results were not statistically different. We found higher risk in persons who were non-Hispanic White or non-Hispanic Black, ≥65 years, less educated (<12 years), never married, and widowed. For O_3, we observed similar patterns with slightly higher risk in males, persons ≥65 years, and those who were less educated (<12 years).

We conducted additional analysis to assess combined disparities in the associations between air pollution and total mortality by combinations of individual- and community-level characteristics (Table 5). We

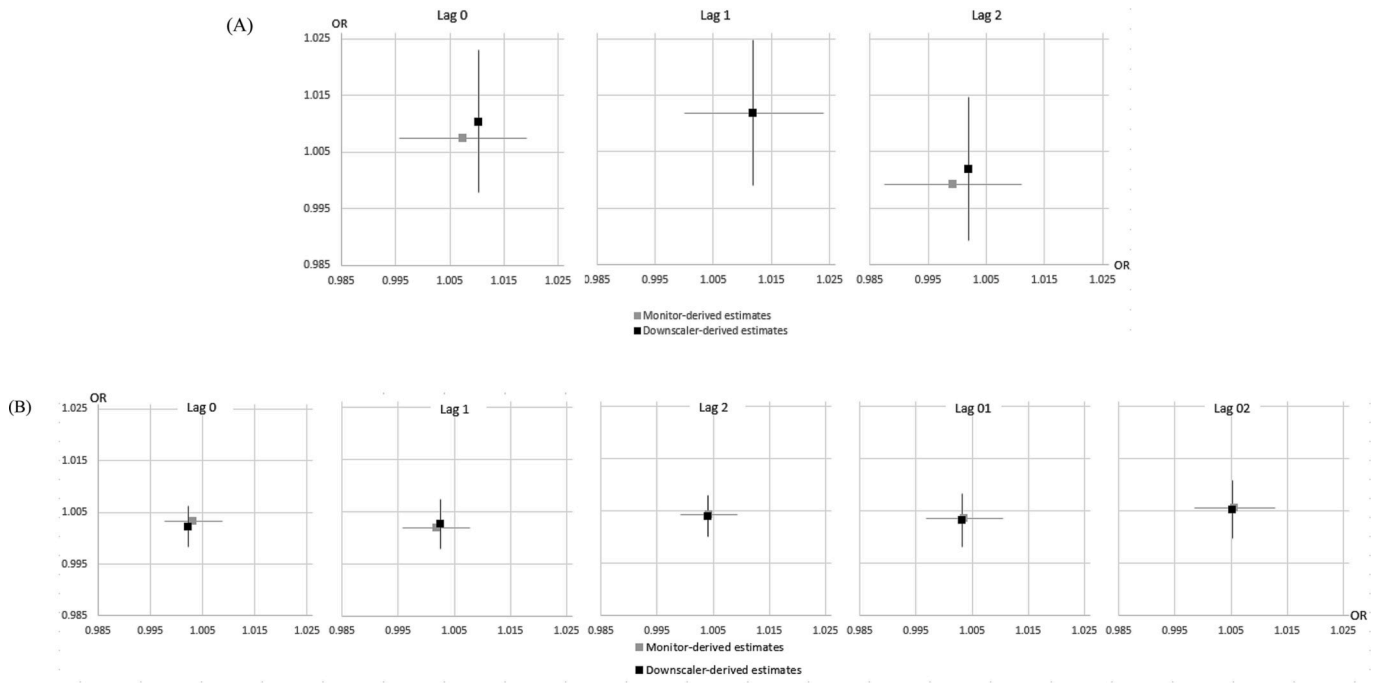

Fig. 2. Downscaler-vs. monitor-derived air pollution effect estimates based on the times and locations for which exposure estimates are available from both methods: (A) $PM_{2.5}$ (B) warm-season O_3. Lines reflect 95% intervals, horizontal estimates represent monitor-derived estimates, Vertical estimates represent downscaler-derived estimates.

Table 3
Association between air pollution and total mortality, stratified by community-level environmental factors.

	$PM_{2.5}$	O_3
Green space		
Average NDVI <0.61	1.020 (1.011, 1.028)	1.006 (1.000, 1.011)
Average NDVI ≥0.61	1.018 (1.009, 1.027)	1.006 (1.001, 1.012)
Blue space (proximity to water bodies)		
<10.2 km	1.017 (1.008, 1.026)	1.004 (0.999, 1.009)
≥10.2	1.020 (1.011, 1.029)	1.008 (1.002, 1.013)
Urbanicity		
urban area ≥50,000 people	1.021 (1.015, 1.028)	1.006 (1.002, 1.010)
urban cluster of 10,000–49,999	1.001 (0.985, 1.018)	1.004 (0.994, 1.015)
urban cluster of <10,000	1.014 (0.890, 1.155)	1.040 (0.965, 1.121)
Census-tract median income		
<41,500 USD	1.021 (1.012, 1.030)	1.004 (0.999, 1.009)
≥41,500	1.016 (1.008, 1.025)	1.008 (1.002, 1.013)

$PM_{2.5}$ lag 01; O_3 lag 02.
Cutoff for green space, blue space, and median income: 50% median.

Table 4
Association between air pollution and total mortality, stratified by individual-level factors.

Characteristics	$PM_{2.5}$	O_3
Sex		
Male	1.023 (1.014, 1.032)	1.008 (1.003, 1.014)
Female	1.015 (1.006, 1.023)	1.004 (0.999, 1.009)
Race/ethnicity		
Non-Hispanic White	1.020 (1.013, 1.027)	1.006 (1.001, 1.010)
Non-Hispanic Black	1.017 (1.004, 1.031)	1.008 (0.999, 1.016)
Hispanic	1.003 (0.931, 1.081)	0.959 (0.917, 1.003)
Non-Hispanic Asian	0.945 (0.859, 1.040)	1.024 (0.967, 1.085)
Non-Hispanic Other	1.018 (0.947, 1.094)	1.017 (0.974, 1.061)
Age at death		
<65 years	1.009 (0.996, 1.021)	1.005 (0.997, 1.012)
≥65 years	1.022 (1.015, 1.029)	1.006 (1.002, 1.011)
Education		
<12 years	1.025 (1.015, 1.035)	1.007 (1.001, 1.013)
High school graduate	1.016 (1.005, 1.027)	1.003 (0.997, 1.010)
1–4 years of college	1.013 (1.000, 1.027)	1.005 (0.997, 1.013)
5 or more years of college	1.017 (0.988, 1.046)	1.013 (0.995, 1.030)
Unknown	1.003 (0.956, 1.052)	1.013 (0.983, 1.044)
Marital status		
Never married	1.024 (1.004, 1.044)	1.009 (0.997, 1.021)
Married	1.017 (1.007, 1.027)	1.006 (1.000, 1.012)
Widowed	1.023 (1.013, 1.033)	1.007 (1.001, 1.013)
Divorced	1.006 (0.988, 1.024)	1.000 (0.989, 1.011)
Unknown	1.123 (0.967, 1.303)	1.077 (0.981, 1.183)

assessed mortality disparities by combinations of race/ethnicity and census-tract median income level. The highest and most significant association between $PM_{2.5}$ exposure and total mortality was found in non-Hispanic Black participants living in areas with the lowest community-level SES. Of non-Hispanic Black participants, a significant association between $PM_{2.5}$ exposure and total mortality was observed only for those living in the lowest census-tract median income level.

4. Discussion

In this study, we evaluated which subpopulations are vulnerable and which factors affect disparities in associations between exposure to air pollution and risk of mortality. Although the results were not statistically different among groups, some factors such as age, education, and urbanicity were associated with higher risk of total mortality from $PM_{2.5}$ exposure. For combinations of individual- and community-level factors, the magnitude of health disparities observed was more pronounced for Non-Hispanic Blacks living in lower community-level SES.

Our findings of positive associations between short-term exposure to $PM_{2.5}$ or O_3 and mortality are consistent with those of many studies in the literature, with similar range of effect size (Supplementary Table 8). As an example, a recent study by Wu et al. (2019) reported that increased exposure to particulate matter ($PM_{2.5}$, PMcoarse, and PM_{10}) in Lanzhou, an industrial city in China, was associated with higher risk of cardiovascular mortality. Other studies observed associations between short-term exposure to $PM_{2.5}$ and total mortality (Li et al., 2017; Yorifuji et al., 2016). Chen et al. (2017) found strong evidence that short-term

Table 5
Association between $PM_{2.5}$ exposure and total mortality in urban areas, stratified by combinations of factors.

	Census-tract median income <33,750 (25%)	Census-tract median income 33,750–41,500	Census-tract median income 41,500–52,269	Census-tract median income ≥52,269 (75%)
Race/ethnicity				
Non-Hispanic White	1.021 (1.003, 1.040)	1.025 (1.009, 1.041)	1.021 (1.007, 1.036)	1.022 (1.008, 1.035)
Non-Hispanic Black	1.035 (1.013, 1.058)	1.006 (0.976, 1.036)	1.008 (0.974, 1.043)	1.020 (0.984, 1.058)
Hispanic	1.005 (0.867, 1.166)	0.926 (0.777, 1.103)	1.106 (0.948, 1.286)	0.971 (0.840, 1.123)
Non-Hispanic Asian	0.978 (0.764, 1.254)	0.867 (0.687, 1.093)	0.986 (0.809, 1.202)	0.906 (0.780, 1.053)
Non-Hispanic Other	1.033 (0.937, 1.139)	1.171 (0.992, 1.383)	0.772 (0.587, 1.015)	0.896 (0.672, 1.195)

exposure to O_3 is significantly associated with increased total mortality.

Our findings on the disparities in air pollution–health associations by some individual- and area-level characteristics are consistent with those of previous studies, which find disproportionate health burdens from air pollution. Many studies showed evidence that some factors such as older age, low education, and living in urban areas are associated with higher risk of mortality from air pollution exposure, consistent with our findings (Bravo et al., 2016; Deguen and Zmirou-Navier, 2010; Son et al., 2012). Wong et al. (2008) suggested that people residing in socially deprived communities have higher mortality risk from ambient air pollution. On the other hand, results for effect modification by some factors have varied. Some previous studies found no differences by sex (Ren et al., 2010), while others found higher effect for males (Chen et al., 2010; Son et al., 2012) or females (Kan et al., 2008; Zanobetti and Schwartz, 2000).

In this study, we did not find significant differences by residential environmental factors such as green and blue spaces. Studies on health disparities attributable to air pollution by residential environmental factors such as residential greenness are limited although some research examined the direct effect of greenness on health outcomes. A few recent studies on effect modification of the $PM_{2.5}$ mortality association found inconsistent results. A recent study by Yitshak-Sade et al. (2019) reported that estimated $PM_{2.5}$ effects on cardiovascular mortality were attenuated by higher neighborhood greenness in areas with lower socioeconomic status. Another study found positive modification of greenness on the $PM_{2.5}$ and mortality association (Kioumourtzoglou et al., 2016). Heo and Bell (2019) found that the association between short-term exposure to particulate matter and hospitalization was lower in areas with more green space. Possible mechanisms of how green space might influence health include reduced risks of physical and mental illnesses by increased opportunities for physical activity and other pathways. Moreover, living near green space may benefit health by facilitating social interaction, and can promote recovery from stress (Richardson et al., 2010). Also, proximity to water bodies may reduce exposure to many urban stressors and have beneficial effects on physiological systems that integrate stress response through higher exposure to health promoting factors and behaviors (Crouse et al., 2018).

Previous findings on disparities in mortality risk related to air pollution were inconsistent across different study areas and populations. The patterns of disparities varied depending on the health outcomes and measures of several variables studied. The differences in health disparities we observed may result from several factors such as variation in population characteristics, distribution and/or composition of characteristics and their interactions within groups (e.g., age, education, and racial/ethnic composition in urban/rural population), biological and generic vulnerabilities, access to health care and quality, social and physical environment, and health-related behaviors (Thomson et al., 2006). In the analysis of combined disparities by race/ethnicity and census-tract median income level, we found that Non-Hispanic Blacks living in lower community-level SES (below median) had the highest risk estimate for the association between $PM_{2.5}$ and total mortality. Our findings indicate that health disparities may relate to socioeconomic differences between/within racial groups; analysis of racial/ethnic differences without consideration of other factors such as socioeconomic status and access to health care may not fully capture the full and complex system. Race/ethnicity and socioeconomic status may be linked through psychosocial pathways such as perceived stress, biological markers of chronic stress (Morello-Frosch et al., 2011; Goodman et al., 2005; Gee and Payne-Sturges, 2004). In general, racial minorities tend to have lower socioeconomic status, however, socioeconomic differences do not fully explain racial disparities. Race/ethnicity is highly correlated with residential location. Poorer neighborhoods tend to have higher rates of psychosocial stressors, which may contribute to health disparities.

A previous study conducted within-race analyses, finding that most of the apparent differences in air pollutant effects found across races were explained by socioeconomic and/or health care disparities (Gwynn and Thurston, 2001). Ito and Thurston (1996) found that black females had the highest risk for air pollution impacts for total, respiratory, and cancer mortality in race and sex-specific analysis. This may relate to multiple factors for the subpopulations and their interactions (e.g., correlation between SES and race/ethnicity at the community level, relationship between percentage of racial minorities living in urban areas with higher levels of pollution and/or harmful residential environment, existing health conditions or behaviors) (Martenies et al., 2017). A challenge to the study of disparities in health risk is that many of the characteristics of interest are often correlated. These complexities change the isolation of responsible factors that contribute to health disparities and different impacts of mechanisms on various populations. Evaluation of disparities in health risk relate to multiple relationships among possible disparity factors. Thus, more research at the local scale is needed to consider the complex interactions among factors on health risk from air pollution exposure.

There are several limitations to this work. We used downscaler predictions of air pollution levels that allow us to estimate air pollution concentrations at locations and time periods without monitors. Although we confirmed that the downscaler-derived findings were robust in comparison to results generated using monitoring data for areas and times with monitors, we could not evaluate the effect estimates in areas without monitors (e.g., non-urban areas). Urban and non-urban areas may have different characteristics of exposure (e.g., pollutant mixtures, chemical compositions) and demographics. Thus, further research considering uncertainty on differences in urban and non-urban areas is warranted. We used 2010 Census data to estimate population characteristics for the study period (2002–2013). Using 2010 Census data may not perfectly reflect actual population characteristics for the period 2002–2013. However, assigning more timely data may introduce uncertainty as well due to some issues such as data from different sources, boundary changes over time. For example, ACS and Census data are not equivalent and there are some differences between the ACS and Census data such as residence rules, reference periods, definitions, and methods between the two data sources that can impact comparability. Thus, we used only 2010 Census data for the whole study period for consistency. Also, for our Census variables there is likely to be little relative change across the Census tracts overall across time. Some measures of disparity factors we considered (e.g., census-tract median household income for community-level socioeconomic status) may not fully reflect the actual aspects of each factor, although the correlations

between several measures of SES in this study were highly correlated with each other (Supplementary Table 9). Many studies have used several measures such as individual- or community-level measures of income and education to represent SES, however SES has complexities of several correlated factors (e.g., historical income) that may affect the associations and combinations of these variables (Williams et al., 2010).

Strengths of our study include the use of geocoded individual-level mortality data with high spatial and temporal resolution exposure data. Our study was able to estimate the health effect of cumulative short-term exposure to $PM_{2.5}$ in areas and time periods without daily monitoring data. For some factors, we were able to assess multiple disparities (e.g., race and SES), which contributes to a better understanding of interactions of disparity factors and to environmental justice more broadly.

5. Conclusions

We provide additional evidence confirming previous work indicating that short-term exposure to $PM_{2.5}$ and O_3 are positively associated with increased risk of mortality. Our assessment of combined disparities indicate that the multiple aspects of disparity factors may affect disproportionate mortality burdens from air pollution exposures. The findings from our work have important implications for environmental decision making by identifying priorities for policy intervention on modifiable factors. This work can help focus on more efficient policy actions to mitigate health impacts for vulnerable populations with limited resources. Our findings on environmental health disparities provide valuable evidence for decision makers and help inform future research on environmental justice.

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Declaration of competing interest

None.

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Appendix A. Supplementary data

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