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Independent and joint contributions of economic, social and physical environmental characteristics to mortality in the Detroit Metropolitan Area: A study of cumulative effects and pathways

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Abstract

Objective: Previous studies have demonstrated associations between race-based residential segregation, neighborhood socioeconomic and physical environmental characteristics, and mortality. Relatively few studies have examined independent and joint effects of these multiple neighborhood characteristics and mortality, including potential mediating pathways. In this study we examine the extent to which associations between race-based residential segregation and all-cause mortality may be explained by multiple socioeconomic indicators and exposure to air pollutants.

Methods: Drawing on data from multiple sources, we assessed bivariate associations between race-based residential segregation (operationalized as percent non-Hispanic Black), education (percent with graduate equivalency degree), poverty (percent below poverty), income inequality (GINI coefficient) and air pollution (ambient PM2.5) and age adjusted all-cause, all race mortality (henceforth all cause mortality) at the census tract level in the Detroit Metropolitan Area. We used inequality curves to assess the (in)equitable distribution of economic and environmental characteristics by census tract racial composition. Finally, we used generalized estimating equations (GEE) to examine independent and joint associations among percent NHB, education, income inequality, and air pollution to all-cause mortality, and test for mediating effects.

Declarations of interest: None

Results: Bivariate associations between racial composition, education, poverty, income inequality, PM2.5 and all-cause mortality were statistically significant. Census tracts with higher concentrations of NHB residents had significantly lower educational attainment, higher poverty, and greater exposure to PM2.5. In multivariate models, education, income inequality and PM2.5 fully attenuated associations between racial composition and all-cause mortality.

Conclusions: Results are consistent with the hypothesis that race-based residential segregation is associated with heightened all-cause mortality, and that those effects are mediated by education, income inequality, and exposure to air pollution at the census tract level. Public health and cross-sector interventions to eliminate race-based residential segregation or to eliminate the maldistribution of educational and economic resources, and environmental exposures, across census tracts could substantially reduce regional inequities in all-cause mortality.

Keywords

Race-based residential segregation and mortality; joint effects of socioeconomic status and physical environment on mortality; Cumulative risk

INTRODUCTION

Race-based residential segregation has been inversely associated with economic and educational opportunities (Logan, Minca, & Adar, 2012; Massey, Gross, & Eggers, 1991; S. R. Reardon, Fox, & Townsend, 2015) and positively associated with air pollutant levels, proximity to hazardous waste facilities and other hazardous land uses (Jones et al., 2014; L. J. Rice et al., 2014). The unequal distribution of multiple risk factors associated with adverse health outcomes has contributed to interest in examining how these risks combine or accumulate to shape health outcomes across geographic areas (Sexton & Linder, 2011; Solomon, Morello-Frosch, Zeise, & Faust, 2016). Much of the literature examining the accumulation of risks conceptualizes social and economic indicators as indicators of population vulnerability, defined as population characteristics associated with heightened probability of exposure to physical environmental threats, or as population susceptibility, defined as characteristics associated with greater health impacts at any given level of exposure (Callahan et al., 2003; Moretto et al., 2016). Race-based residential segregation in the context of the United States is conceptualized here as an indicator of the social environment, reflecting historical as well as contemporary policies that inequitably shape access to housing across geographic areas. Segregation, in turn, shapes access to resources such as education and employment opportunities, as well as the ability to avoid exposures that are inimical to health, such as air pollution (Phelan & Link, 2015; Schulz, Williams, Israel, & Lempert, 2002; Williams & Collins, 2001; Williams & Mohammed, 2013). Relatively few studies have examined the combined implications for health of these multiple pathways through which segregation may impact health outcomes. In this paper, we examine the independent and joint contributions of race-based residential segregation, indicators of the economic environment, and exposure to air pollutants to all-cause mortality, in the Detroit Metropolitan Area (DMA).

Conceptual Framework

In the century since W.E.B. DuBois documented the physical and social environments in which "the Philadelphia Negro" lived, worked and sought to maintain health and well-being (Du Bois, 1899), a body of work has emerged examining associations between neighborhood racial composition, neighborhood economic and physical environmental characteristics and health. Building on prior models of segregation and health (Schulz et al., 2016, 2002; Williams & Collins, 2001), we conceptualize racial residential segregation as a manifestation of racist ideologies that shape health outcomes through multiple pathways, encompassing both health protective and health eroding impacts. These include systematic impacts on the social, economic and physical contexts in which people live, shaping opportunities for health. Below we briefly review the evidence linking racial or ethnic composition of neighborhoods, as one indicator of segregation, with socioeconomic and physical environmental contexts, and with all-cause mortality.

Race-based residential segregation.—Racial residential segregation can be conceptualized as an indicator of historical and contemporary social and political environments, and as one manifestation of ideologies of white supremacy, a political, economic and cultural system that rests upon the systematic devaluation of Black and Brown lives (Ansley, 1997). Historically in the U.S., housing policies and practices explicitly created and reinforced racial segregation (Rothstein, 2017), assuring separation of non-Hispanic Whites (NHW) from racialized groups (Charles, 2003; Rothstein, 2017). The Fair Housing Act of 1968 provided protections against housing discrimination on the basis of race, color, religion or national origin. However, racial segregation has remained high in many areas of the country, in particular for non-Hispanic Black (NHB) Americans (Reibel & Regelson, 2011). The geographic separation of NHB Americans from, in particular, NHW is reflective of, and reinforces, deeply embedded ideologies of white supremacy that structure social relationships in the U.S. (Kendi, 2017; Rothstein, 2017).

Socioeconomic inequalities.—There is substantial evidence that residential racial segregation contributes to economic divestment, limited educational and employment opportunities, as well as foreclosure risk (Bell & Lee, 2011; Morello-Frosch & Jesdale, 2006; Rugh & Massey, 2010; Sugrue, 2014). Socioeconomic characteristics of neighborhoods, including opportunity structures, economic resources and income inequalities, are associated with health inequities. Across various measures of socioeconomic position (SEP), incremental increases in social standing largely correlate with improved community health outcomes (Andersen et al., 2018; Meijer, Röhl, Bloomfield, & Grittner, 2012). NHBs and Hispanics in the United States are disproportionately likely to reside in communities with reduced access to quality education and employment opportunities (Logan et al., 2012; Massey et al., 1991; S. R. Reardon et al., 2015) and to benefit less in terms of occupational and economic returns on educational attainment (Phelan & Link, 2015; Williams & Mohammed, 2013). Several studies have linked neighborhood level characteristics such as educational attainment and economic indicators to racial and ethnic health inequities (Denney, Saint Onge, & Dennis, 2018; LaVeist, Pollack, Thorpe, Fesahazion, & Gaskin, 2011; Mode, Evans, & Zonderman, 2016; Yao & Robert, 2011). Lower economic status contributes to adverse health effects through

multiple mechanisms including, for example, poor quality housing (Camacho-Rivera, Kawachi, Bennett, & Subramanian, 2014; Evans & Kantrowitz, 2002; Evans & Kim, 2010) and reduced access to healthcare (Kirby & Kaneda, 2005). Residents of areas with limited economic opportunities and access to resources (e.g., transportation, health and social services) which can buffer adverse effects of poverty (Dinwiddie, Gaskin, Chan, Norrington, & McCleary, 2013; Haley et al., 2017; Kirby & Kaneda, 2005; Meijer et al., 2012; White, Haas, & Williams, 2012) face particular challenges.

Physical environment.—Exposure to ambient air pollution is an important risk factor in the physical environment. It is associated with increased risk of multiple adverse health outcomes, including cardiovascular mortality (Brook et al., 2010; Jerrett et al., 2013; Ostro et al., 2015), respiratory hospitalization (Neupane et al., 2010), asthma exacerbation (Fan, Li, Fan, Bai, & Yang, 2016; Li et al., 2011; Strickland et al., 2010), incidence and duration of respiratory symptoms (Johannson et al., 2014; Stern et al., 2013), declines in lung function (Gehring et al., 2013; M. B. Rice et al., 2013; Urman et al., 2014), preterm delivery and low birth weights (Pedersen et al., 2013; Shah, Balkhair, & Knowledge Synthesis Group on Determinants of Preterm/LBW Birth, 2011; Stieb, Chen, Eshoul, & Judek, 2012), and restricted activity (Fann et al., 2012; Nam, Selin, Rielly, & Paltsev, 2010). Fine particulate matter ($PM_{2.5}$) is more strongly associated with morbidity and mortality than coarse particulate matter (PM_{10}) (U.S Environmental Protection Agency, 2002). Adverse health effects are observed at pollution levels below current U.S. National Ambient Air Quality Standards (NAAQS) and common to many U.S. cities (Lepeule, Laden, Dockery, & Schwartz, 2012; Shi et al., 2016).

Combined socioeconomic and physical environmental factors and health.—In addition to the social and economic vulnerabilities described above, NHB and Hispanic communities, and those with low to moderate incomes, are disproportionately exposed to air pollutants (Clark, Quinn, Dodge, & Nelson, 2014; Collins, Grineski, Chakraborty, Montgomery, & Hernandez, 2015; Gray, Edwards, & Miranda, 2013; Huang & London, 2012; Jones et al., 2014; Mohai, Lantz, Morenoff, House, & Mero, 2009; Schulz et al., 2016). Low to moderate economic status contributes to challenges avoiding exposure and may exacerbate adverse health effects of exposures (Sadd, Pastor, Morello-Frosch, Scoggins, & Jesdale, 2011). Mechanisms include, for example, poorer quality housing (Adamkiewicz et al., 2014; Jacobs, 2011) and reduced access to health care (Forno & Celedon, 2009). Children and older adults are more susceptible to adverse health effects of exposure (Bell & Lee, 2011; Kelishadi & Poursafa, 2010; National Environmental Justice Advisory Council, 2014; Sacks et al., 2011; Solomon et al., 2016). Yet, relatively few studies have examined combined effects of exposure to air pollution in conjunction with population social and economic vulnerabilities, or quantified the health impacts associated with reductions in air pollution in communities as these may vary with characteristics of exposed populations. Using data from the Detroit Metropolitan Area (DMA), we examine the independent and joint contributions of air pollution exposure and social and economic characteristics of neighborhoods to all-cause mortality. Despite evidence linking racial residential segregation to circumscribed access to resources necessary to maintain health (e.g., educational opportunity, economic opportunity) and to excess exposure to environments detrimental to

health (e.g., air pollutants), results from studies examining associations between neighborhood racial composition and mortality have been mixed. In a recent review article, Bécares and colleagues (2012) found that several studies reported positive associations between percent black residents and all-cause mortality (Erwin, Fitzhugh, Brown, Looney, & Forde, 2010; Jackson, Anderson, Johnson, & Sorlie, 2000; LeClere, Rogers, & Peters, 1997), while others reported null or negative associations (Blanchard, Cossman, & Levin, 2004; Hutchinson et al., 2009; Inagami et al., 2006).

There are a number of potential explanations for these mixed results. The reported findings may be influenced by differences across studies in the extent to which they account for potential mediating pathways as well as neighborhood level correlations between racial composition, restricted economic and educational opportunities, and adverse physical environmental exposures (Huang & London, 2012; Sadd et al., 2011; Schulz et al., 2018, 2016; Su et al., 2009). Researchers have sought increasingly to examine joint effects of physical environmental exposures with social and economic characteristics on health (Collins et al., 2015; Mohai et al., 2009) in order to understand unique and joint effects of these correlated pathways. To the extent that adverse physical environmental exposures cluster in communities with limited social and economic resources, effects may accumulate, compounding implications for racial and socioeconomic health inequities (Bell & Lee, 2011; Callahan et al., 2003; Commission on the Social Determinants of Health, 2008; Dankwa-Mullan et al., 2010; Kelishadi & Poursafa, 2010; Mujahid et al., 2008; Sacks et al., 2011; Solomon et al., 2016).

Understanding joint effects of multiple, often linked, social, economic and physical environmental factors thus remains an important challenge (Alvarez, Appleton, Fuller, Belcourt, & Kubzansky, 2018; Huang & London, 2012; Sadd et al., 2011; Su et al., 2009). This research examines hypothesized relationships between multiple area level characteristics and all-cause mortality, using specific measures that may be relevant to developing local policies, to address this important gap (Markevych et al., 2017).

Overarching goal.—The primary research question addressed in this paper is the extent to which area level social, economic and physical environmental characteristics contribute to geographic variations in mortality in the DMA. Relationships are modeled at the census tract level to examine contributions to census tract level all-cause, all race mortality. Understanding the factors that drive the racial and socioeconomic patterning of risk within and across the DMA, including processes through which cumulative advantage and disadvantage accrue, is essential to understand the drivers of poor health outcomes and health inequities and to address them. Among these potential pathways are synergistic effects of the physical environment with social and economic characteristics.

Context.

Historically, the DMA, made up of Macomb, Oakland, and Wayne Counties, has been among the most racially segregated regions in the nation (Farley, 2011; Fry & Taylor, 2012). Numerous factors contributed to this segregation, including housing agreements prominent prior to 1948 that restricted home-ownership in areas of the city of Detroit to whites only

(Sugrue, 2014). Despite Detroit's major contributions to the Civil Rights Movement, discrimination in the real estate market, including federally-backed home mortgages available to NHWs but not NHBs, perpetuated segregation and reduced NHBs' access to suburban employment opportunities (Farley, 2015; Farley, Danziger, & Holzer, 2000; Sugrue, 2014). Today's DMA reflects these historical and contemporary processes (see Figure 1). NHB populations in Wayne, Oakland, and Macomb counties are clustered in older, urban areas ("2015 Data Release New and Notable," 2016). Eighty-five percent of Wayne County's NHB population is located in Detroit city ("2015 Data Release New and Notable," 2016). Detroit city is also home to an Hispanic community whose roots extend back nearly a century (Rodriguez, Bowie, Frattaroli, & Gielen, 2009). NHBs and Hispanics across the DMA are disproportionately represented in households below the federal poverty line (Jargowsky, 2015). Over 57% of Detroit's children (Annie E. Casey Foundation, 2015; New Detroit Coalition, 2014), compared to 37%, 20%, and 13% of children in Wayne, Macomb, and Oakland counties, respectively, live in households below the poverty line ("2015 Data Release New and Notable," 2016).

DMA census tracts with greater concentrations of NHB and Hispanic residents, households below the poverty line, and residents who have not completed high school are more likely to have higher levels of exposure to diesel PM (a component of air pollution more broadly) and associated cancer and respiratory health risks (Schulz et al., 2016). This study extends previous research in the DMA by examining the independent and joint associations between racial composition, physical environmental exposures (fine particulate matter or $PM_{2.5}$), social and economic characteristics (education, poverty, income inequality) and all-cause mortality in the DMA.

DATA AND METHODS

The study was conducted as part of Community Action to Promote Healthy Environments, a community-based participatory research partnership designed to equitably engage community, academic, and health service provider organizations in research, and to translate results from that research into action to promote health equity (see Acknowledgments). The University of Michigan granted institutional review board approval for this study on 24 January, 2013.

Data.

Data for the analyses described below were drawn from multiple sources. Demographic data were drawn from the 2009–2013 American Community Survey, aggregated over a 5-year period in keeping with ACS recommendation (U.S. Census Bureau, 2018, 2020). Mortality data were provided by the Michigan Department of Health and Human Services (MDHHS) and include records of all deaths that occurred in the Detroit metropolitan area (Wayne, Oakland, and Macomb Counties) between the 2009–2013 period. Information on fine particulate matter was drawn from the EPA's National-Scale Fused Air Quality Surfaces Using Downscaling Tool and the Community Multiscale Air Quality (CMAQ) model, for the calendar years 2009–2013. The downscaler model combines air quality monitoring and modeling data to provide better fine-scale predictions of air pollutant levels at local and

community scales (Berrocal, Gelfand, & Holland, 2010b, 2010a, 2012). Tests of this model indicate that predictions are better calibrated and offer improved predictive intervals compared with either Bayesian melding or ordinary kriging (Berrocal et al., 2010b). Additional information about the construction of CMAQ data is available from the EPA website (https://www.epa.gov/air-research/downscaler-model-predicting-daily-air-pollution): It is downloadable at the census tract level.

Measures.

All measures are constructed at the census tract level.

Dependent variable: Age adjusted all-cause mortality at the census tract level. This variable is calculated using mortality data obtained from the MDHHS, as described above, and including all deaths from the DMA area during the period 2009–2013. Deaths were geocoded and linked to the census tract in which the decedent resided. Age adjusted all-cause mortality was calculated for each census tract by calculating age-specific mortality within the tract and multiplying it by the proportion of the total population in that age group, then summing across all age groups for each census tract (Curtin & Klein, 1995). This method adjusts for the different age structures of populations across census tracts. Age adjusted all-cause mortality is reported as deaths per 100,000 population. Because deaths are rare events, they can fluctuate over time: using broader periods of time will allow for more stable estimates. Therefore, we used the five-year average (2009–2013) to calculate the 2013 mortality rate at the census tract level (IOM, 1997), aligned with multi-year averaging used by prior ecological studies of area mortality (Erwin et al., 2010; Hutchinson et al., 2009).

Independent variables are listed here.—*Percent Non-Hispanic Black* (NHB), derived from American Community Survey (ACS) data (2009–2013), was measured as the percent of the total population in each census tract that identified as non-Hispanic Black or African American, which was used as a proxy for race-based residential segregation. *Education* was assessed as the percent of census tract residents aged 25 and older with less than high school education or Graduate Equivalency Degree (GED), drawing on ACS data, 2009–2013. *Income* was assessed as the percent of households in a census tract with incomes below the federal poverty level, 2009–2013 (Mooney, Richards, & Rundle, 2014). *Income inequality* was measured using the GINI coefficient (Kondo et al., 2009), derived from the ACS at the census tract level, 2009–2013 (U.S. Census Bureau, n.d.). *Air pollution* was assessed as $PM_{2.5}$ concentration, calculated at the census tract level, using 2009–2013 $PM_{2.5}$ 24-hour annual average concentrations.

For each of the census tract level independent variables above, models were run using continuous variables, and also using scores calculated based on their percentile distribution, in quintiles ranging from 1 (<P20) to 5 (>P80). Each tract in the study area was rank ordered, and assigned a quintile score for each of the above variables using this ranking system. Quintiles were entered into the models using Q1 as the referent: A positive coefficient indicates that the value for the quintile in question is higher than for the referent, and a negative coefficient indicates that the value is lower than the referent. Results presented use the rank ordered quintiles, which allow for non-linear associations.

Analysis.

To estimate the extent to which education, poverty, income inequality and $PM_{2.5}$ are (in)equitably distributed across census tracts by percent NHB, the cumulative proportion of the population, ordered by area-based percentages of racial concentration was plotted against the cumulative share of poverty, education, income inequality and $PM_{2.5}$, respectively. In the case for which each population group has the same share of the impact of the environmental hazard or vulnerability, the curve coincides with the equality line. If the curve lies above the equality line, the inequality index is negative, indicating that more disadvantaged groups encounter greater environmental exposure or vulnerability burdens. If the curve lies below the equality line, then more advantaged groups carry a higher proportion of cumulative environmental exposure or vulnerability burdens. A summary measure of inequality is defined as twice the area between the curve and the equality line:

$$I = 1 - 2\int_1^n e(s)\,ds$$

This measure gives a quantitative summary of inequality among groups. In order to assess the integral we used the “trapezoidal rule” (Atkinson, 1989) to approximate the region under the graph of the function e(s). The value of 0 is the lowest level of inequality where all groups have the same exposure to the variable of interest. When the inequality score is negative, it indicates that less advantaged groups bear a disproportionate burden of exposure: The highest level of inequality, where disadvantaged groups bear the burden of all the exposure is −1 (Kakwani, Wagstaff, & van Doorslaer, 1997).

To examine the independent and joint associations of indicators of social, economic and physical environments to all-cause mortality at the census tract level, we used the generalized estimating equation (GEE) modeling approach with identity link and exchangeable correlation matrix (Hardin & Hilbe, 2003; Liang & Zeger, 1986). This approach allows us to somewhat control the spatial clustering of the outcome with the appropriate selection of the block-exchangeable correlation matrix and thus any possible spatial correlations were then taken into account (Hardin & Hilbe, 2003; Zeger, Liang, & Albert, 1988).

Different model configurations were considered to assess the individual and multivariate associations between each independent variable and all-cause mortality. Models examine single-social factor-response and multi-social factor-response relationships. To assess the most correct specification of the models, in addition to additive models, we considered multiplicative models assessing interactions between independent variables data (Maddala & Lahiri, 2009). Although there were some statistically significant ($p < 0.05$) interaction terms, they were few in number and patterns were inconsistent. Based on these results, and assessment of the goodness of fit statistics (QIC and BIC), we were unable to conclude that the interaction models were a better fit than the main effect models. As a result, the final models reported in this paper report main effect models. To assess the sensitivity of models to varying specifications of the variables, we ran models using the rank-ordered quintile versions of each variable as described above and their binary versions using the third quartile

as the threshold or cut point. Patterns were similar: Findings are reported using the rank-ordered quintile versions, 95% confidence intervals, and p-value for each coefficient of interest.

RESULTS

Table 1 shows descriptive statistics for each of the indicators included in the models. At the census tract level, the mean proportion of residents who identified as NHB was 29.7 (SD=36.9). The mean proportion of residents 25 and older with less than a high school education was 14.1 (SD=10.2). The mean proportion of households with incomes below the poverty line was 17.9 (SD=15.4), and the mean annual level of $PM_{2.5}$ for census tracts in the DMA was 10.2 μg/m^3 (SD = 0.4). The age-adjusted mean number of deaths per year at the census tract level was 648.5 (SD=235.9).

As shown in Model 2, all-cause mortality rates were significantly greater for those residing in census tracts with larger proportions of adults who did not have a high school diploma, households below the poverty line (Model 3), and greater income inequality (Model 4), respectively. For education and income inequality, odds of mortality were significantly higher in each of quintiles 2–5 compared with mortality in the referent (lowest) quintile. For poverty, those in the 3rd (B=63.5, p=0.003) and 4th (B=62.6, p=0.003) quintiles showed comparable increases in all-cause mortality rates, while those in the 5th quintile experienced rates of all-cause mortality that were 114.6 per 100,000 population greater than those in the lowest poverty quintile (p=0.000).

Finally, for $PM_{2.5}$, residents of census tracts in the second (B=142.6, p=0.000), third (B=156.1, $p = 0.000$), fourth (B= 183.2, $p = 0.004$), and fifth (B=206.5, $p < 0.001$) quintiles experienced significantly higher rates of all-cause mortality compared to residents of census tracts in the first quintile.

Table 3 presents results from analyses of inequality indices. Census tracts with higher percentage of NHB residents are significantly more likely to have a higher proportion of residents aged 25 and older who have not completed high school, to have a higher proportion of residents living below the poverty line, and to have higher proportions of high $PM_{2.5}$ exposure. A trend toward greater income inequality in census tracts with a greater percentage of NHB residents was not statistically significant.

These results are consistent with prior research that has demonstrated that areas with greater percentages of NHB residents have reduced education (Bennett, 2011; Reskin, 2012), higher rates of poverty (Krieger, Waterman, Gryparis, & Coull, 2015), and exposure to airborne pollutants (Jones et al., 2014; Krieger et al., 2015; Rice et al., 2014). Our next research question assessed the individual and joint contributions of racial composition, socioeconomic position and airborne particulate matter. Models including both poverty and education simultaneously were unstable. Examination of variance inflation factors (VIF) identified multicollinearity concerns for poverty (VIF=4.06). VIF for other variables did not approach levels of concern, with VIF =2.89 for education, and VIF< 2 for percent NHB, income inequality, and $PM_{2.5}$. Thus models for which results are reported in Table 4 include

percent NHB, percent of adults without a high school diploma, income inequality and PM2.5.

Results presented in Table 4 show that initially significant positive associations between percent NHB and all-cause mortality (presented in Table 2 and replicated here in Model 1 for ease of comparison) are attenuated with the inclusion of education, income inequality and air quality variables. In Model 1, mortality rates in census tracts in the 4th (B=40.8, p=.055) and 5th (B=139.9, p<.001) quintiles of %NHB are greater than those in the 1st quintile. After accounting for socioeconomic indicators (percent high school completion, income inequality) in Model 2, mortality rates remain significant higher in census tracts in the 5th quintile for percent NHB (B=68.6, p=.008). In Model 3, which accounts for both socioeconomic and air pollutant indicators, mortality rates in census tracts in the 2nd (B=−29.4, p=0.140), 3rd (B=−22.1, p=0.283), 4th (B=−26.5.3, p=0.120) and 5th (B=32.5, p=0.237) quintile of percent NHB are not significantly different from those in the first quintile.

Associations between education and all-cause mortality are attenuated somewhat from results shown in Table 2, but remain statistically significant for each quintile. Rates of all-cause mortality are significantly higher in census tracts with greater proportions of adults who have not completed high school compared with those in the lowest quintile, as follows: 2nd (B=66.4, p=0.001), 3rd (B=104.6, p<0.001), 4th (B=102.4, p<0.001) and 5th (B=87.3, p=0.002) quintiles. Substantial overlap in the confidence intervals for quintiles 2–5 does not allow distinctions to be drawn across those quintiles.

In the full model, associations between income inequality and all-cause mortality remain in the expected direction, but are attenuated with the inclusion of other variables: 2nd (B=24.5, p=0.162) , 3rd (B=40.0, p=0.044), 4th (B=28.5, p=0.193) and 5th (B=76.7, p=0.002). As noted above, while quintiles 2–5 show rates of mortality that are significantly greater than the referent (quintile 1, lowest level of income inequality), the overlap in confidence intervals across quintiles 2–5 does not warrant distinctions across those quintiles. These results are generally consistent with the hypothesis that greater income inequality is associated with heightened all-cause mortality.

Finally, associations between exposure to $PM_{2.5}$ and all-cause mortality remain statistically significant for each quintile, compared to those in the lowest quintile of exposure. Mortality rates in the 2nd (B=144.6, p<0.001), 3rd (B=129.9, p<0.001), 4th (B=115.2, p<0.001) and 5th (B=131.4, p<0.001) quintiles are significantly greater than those in the first quintile: These effects are evident after accounting for the effects of racial composition and socioeconomic indicators.

A formal test (MacKinnon, Fairchild, & Fritz, 2007) indicates that associations between percent NHB and all cause, all race mortality are fully attenuated in models including census tract level percentage over age 24 who have completed high school education or equivalent, income inequality and $PM_{2.5}$ exposure (p<0.001) (results not shown).

DISCUSSION

Findings reported here indicate that census tract level racial composition, proportion of adults with less than a high school education, proportion of households below the poverty line, income inequality, and exposure to $PM_{2.5}$ are each associated with age-adjusted all-cause, all-race mortality at the census tract level in bivariate models. Census tracts with higher proportions of NHB residents are disproportionately likely to have greater proportions of adults who have not completed high school, households with incomes below the poverty line, and higher levels of exposure to $PM_{2.5}$. After accounting for census tract level indicators of education, income inequality and particulate matter, associations between census tract percent NHB and all-cause mortality are no longer statistically insignificant: Formal tests indicate that racial composition is attenuated in the full model. Associations between education, income inequality and fine particulate matter remain significant predictors of mortality in the full model. We discuss each of these findings below.

Percent non-Hispanic Black.

Our finding that the percent NHB at the census tract level was positively associated with all-cause mortality in bivariate models, and not significantly associated with all-cause mortality in multivariate models joins mixed findings reported previously in the literature (Bécares et al., 2012; Blanchard et al., 2004; Erwin et al., 2010; Hutchinson et al., 2009; Inagami et al., 2006; Jackson et al., 2000), and suggests the importance of guiding theoretical frameworks and controls for potential mediators in multivariate models. Specifically, our findings suggest that associations between percent NHB and all-cause mortality in bivariate models may be explained by heightened exposure to adverse socioeconomic contexts (circumscribed educational opportunities, poverty, income inequality) and physical environmental exposures ($PM_{2.5}$). These patterns are consistent with the conceptual framework guiding this analysis, which suggests that the physical separation of NHBs in geographically distinct neighborhoods contributes to inequities in the distribution of opportunities for education and economic inequalities, shaping access to resources that are essential for the maintenance of health. Furthermore, associations between the racial composition of census tracts and levels of $PM_{2.5}$ also suggest disproportionate exposure among residents of neighborhoods with high concentrations of NHB residents to physical environmental conditions that are inimical to health.

Results reported here suggest that excess mortality associated with the concentration of NHB residents at census tract level is attenuated by circumscribed access to educational opportunities and heightened exposure to air pollution. Previous studies using ecological models have reported mixed results, with some finding positive associations between racial composition and mortality (Erwin et al., 2010) and others reporting inverse associations between racial composition and black mortality (Hutchinson et al., 2009). Our finding of an initially significant association of percent NHB with all-cause, all-race mortality (Table 2) which is attenuated after inclusion of potential mediating pathways (socioeconomic indicators, environmental pollutants) suggests one potential reason for the mixed findings previously reported may be linked to the analytic and conceptual models that inform that

previous work. The inclusion or exclusion of variables that represent potential mediating pathways may influence reported findings.

Furthermore, variations in findings across local areas may also reflect regional and local variations in the patterning of racial segregation and its implications. While ideologies of white supremacy are a national phenomenon, imbedded in national policies and practices, local expressions of those policies and practices, and local community responses to them may contribute to variations in patterns and outcomes. Specifically, associations between percent NHB and health outcomes may vary with the distribution of percent NHB, poverty characteristics (Kershaw et al., 2011), social characteristics of the neighborhood (e.g., social capital, histories of resistance and political organization) (Minkler & Freudenberg, 2010), and access to countervailing identities that may be health protective (Geronimus et al., 2016). These local variations may be obscured through the use of national datasets, as well as local datasets in which key variables may be unavailable, contributing to mixed findings reported in the literature. Close attention to local histories, contemporary political, social and economic dynamics, model specification and theoretical and empirical pathways is critical to development of a more nuanced understanding of these associations and pathways, as they emerge within local contexts. Such analyses can be importantly enhanced by engaging community residents and leaders in the process of framing and interpreting the research findings (Israel, Eng, Schulz, & Parker, 2013). Findings from the cross-sectional analyses reported here are consistent with the proposition that adverse associations between percent NHB and all-cause, all-race mortality in the DMA are mediated through socioeconomic indicators (e.g., education) and physical environmental exposures (e.g., air pollutants). Future studies using longitudinal analyses are warranted to more specifically assess mediation effects.

Socioeconomic Status.

The proportion of the population aged 24 and older with less than a high school education remained significantly associated with all-cause mortality in multivariate models accounting for racial composition, income inequality and $PM_{2.5}$. The direction of associations between income inequality and all-cause mortality remained consistent although effect sizes were substantially attenuated in the full models. It is plausible that the associations between income inequality and all-cause mortality may be at least partially explained by other variables included in the model. For example, income inequality has been linked to widening gaps in educational achievement (Reardon, 2014). Individuals with low socioeconomic status who live in areas with higher income inequality are more likely to attend poorer resourced schools (Reardon, 2014) and drop out of high school, perhaps due to a perception that an investment in education will have a low economic return (Kearney & Levine, 2016). Income inequality has also been associated with lower levels of investment in public educational systems within poorer communities (Reardon, 2014). Such disinvestment in educational systems has been previously reported to be associated with lower educational attainment (Duncan & Murname, 2011) and linked with growing levels of income inequality resulting from this opportunity gap. Similarly, associations between income inequality and pollution have been documented (see Cushing, Morello-Frosch, Wander, & Pastor, 2015 for a recent review), with more equitable states (assessed variously as voter turnout, educational

attainment, energy prices, employment opportunities) associated with reduced pollution (Boyce, Klemer, Templet, & Willis, 1999; Cushing et al., 2015; Templet, 1995). Further examination of associations among the mediating pathways posited in our conceptual model, for which this analysis provides some cross-sectional support, is warranted.

Particulate Matter.

Our finding that $PM_{2.5}$ remains significantly associated with all-cause mortality in the full model is consistent with the hypothesis that $PM_{2.5}$ exerts independent effects on health, above and beyond those captured with the social and economic indicators included in the models presented here. This finding is consistent with the conceptual model that underlies this analysis, which suggests that residents of more segregated neighborhoods are more likely to be exposed to toxic physical environments (in this case, operationalized as $PM_{2.5}$), and that those exposures are associated with all-cause mortality even after accounting for racial composition and socioeconomic indicators. Thus, they suggest that race-based residential segregation operates not only through its impact on social and economic resources such as education and economic opportunities (reflected in income inequalities) but also through additional exposure to risk factors in the physical environment, such as heightened exposure to air pollutants.

Joint effects:

The results described above are derived from multivariate models that assess the joint effects of racial composition, socioeconomic indicators (educational attainment, income inequality) and exposure to air pollution. They begin to address the question of the extent to which these variables, which often cluster spatially, are independently and jointly associated with adverse health outcomes. Results reported here are consistent with the hypothesis that census tracts with higher concentrations of NHB residents experience higher levels of all-cause, all-race mortality. They also indicate that access to educational and economic resources are unequally distributed across census tracts in the DMA, with lower levels of educational attainment and higher levels of poverty in census tracts with higher concentrations of NHB residents. Similarly, exposure to poor air quality is unequally distributed, with higher levels of $PM_{2.5}$ in census tracts with higher concentrations of NHB residents.

Multivariate models suggest that educational attainment, income inequality, and exposure to $PM_{2.5}$ attenuate bivariate associations between racial composition and all-cause, all-race mortality. Further, significant associations between each of these pathways and mortality in the multivariate models suggest that each pathway may help to explain the initial associations between racial composition and mortality. These findings are consistent with the conceptual framework described above, and with literature linking segregation to adverse health outcomes through multiple mediating pathways.

Limitations: There are well known limitations of ecological analyses such as those used here, which examine data aggregated at the census tract level. These include what is commonly termed the ‘ecological fallacy’ in which relationships observed for populations or groups are interpreted as if they are true for individuals within that population (Levin, 2006). Results from the analyses reported here are interpretable as applicable to understanding

population risk of mortality and should not be inferred to individual risk of mortality. In addition, the outcome used in this analysis of all-cause mortality for all racial groups combined does not examine, for example, specific causes of mortality or variations between racial groups on the impact of the factors considered.

Our use of cross-sectional analysis allows us to describe associations, including the attenuation of NHB associations with mortality, but does not allow causal attribution nor formal tests of mediation. The use of longitudinal models to more explicitly assess associations over time, lag times to mortality, and test mediational pathways will be critical next steps to more formally test the conceptual model presented in this paper.

There are several limitations of our use of percent non-Hispanic Black (NHB) as a proxy for race-based residential segregation. First, although it is easily operationalized, this measure does not account for the clustering of racial groups within census tracts (Kramer & Hogue, 2009). For example, a neighborhood that is 80% NHB means something different in a city that is 20% NHB compared to a city that is 79% NHB, such as Detroit. Commonly used measures of segregation such as the dissimilarity index and isolation index assess more specifically the distribution of racial groups within and across neighborhoods. A recent analysis reported significant correlations between percent NHB and black/white isolation, using the isolation index (Yang & Matthews, 2015). A second study reported significant associations between both racial composition and segregation measured using the dissimilarity index, and mortality (Nuru-Jeter & LaVeist, 2011), suggesting that these measures may capture different constructs. A second important criticism of the use of percent NHB is the failure to contextualize the measure within the larger area, that is, as an absolute, rather than a relative indicator of racial segregation. The conversion of raw percent NHB in our measure into an ordinal variable reflecting quintiles of census tract level percent NHB partially addresses this concern. Specifically, the lowest quintile (Q1) of our measure includes the 20% of all census tracts in the DMA with the smallest proportion of NHBs, ranging from 0.0%-1.7% NHB. The highest quintile (Q5) captures the 20% of rank ordered census tracts in the DMA with the highest proportion of NHB residents, ranging from 83.0–100.0% NHB. Thus, findings reported here reflect the associations with mortality of residing in census tracts within the DMA with relatively higher proportions of NHB residents, compared with those census tracts in the lowest 20% of percent NHB in the DMA. Finally, our focus in this paper on percent NHB does not account for effects of segregation of Hispanic or Latinx households in census tracts within the tri-county area. Several studies have found evidence of disproportionate exposure to pollutants among communities with higher proportions of Hispanic residents (Jones et al., 2014; Martenies, Milando, Williams, & Batterman, 2017). Further analyses examining segregation across multiple racial and ethnic groups would importantly extend this analysis.

This analysis used data aggregated over a 5-year period and is cross sectional rather than longitudinal. This methodology does not allow us to capture effects over time or to determine causality. Such temporal effects have been established through previous studies, and the cross-sectional techniques used in this analysis may underestimate health impacts that emerge over time as the effects of exposure accumulate (e.g. associations with cancer, which may become visible over longer time periods than are captured in our analysis). In

that regard, these estimates may be conservative. The strength of the analytic method lies in its ability to capture geographic variation of multiple variables with variant units of analysis, and is described in greater detail in the “Strengths” section that follows.

Given limitations of available data, we are unable to specifically include potentially protective pathways or mechanisms in the models, for example, measure of social capital or social support. Given protective effects of social cohesion reported elsewhere (Bécares, Nazroo, & Stafford, 2009; Hutchinson et al., 2009), future studies assessing positive social relationships as potential pathways linking racial or ethnic density to reduced mortality are critical to understanding local variations in associations between racial composition and health outcomes.

Our measure of PM was derived from downscaler models with estimates linked to census tracts. The use of aggregated data over a five-year period obscures temporal variations in the data that are likely linked to variations in mortality. Modeled data used for these estimates is derived from data captured by monitors at specific locations, and is affected by uncertainty in the estimates. Finally, $PM_{2.5}$ is just one of many potential physical environmental risk factors to which residents of racially segregated communities may be disproportionately exposed. Others include excess exposure to lead through disproportionate older or poorly maintained housing stock, toxics through increased proximity to hazardous land uses, and poor-quality infrastructure (e.g., sidewalk maintenance), documented extensively in the literature. As a result of these limitations in our measure of physical environmental exposures, the findings reported here are likely conservative.

Strengths.

Despite the limitations of ecological studies described above, there is increasing recognition that population level studies play an important role in studying important public health problems, particularly for problems that operate at the population level (Levin, 2006; Pearce, 2000; Susser, 1994). As argued by Geoffrey Rose (2001) in his classic work on the determinants of population rates, as opposed to determinants of individual cases, it is essential to study the characteristics of populations rather than the characteristics of individuals in order to understand mechanisms of intervention with the potential to shift the mean risk for the group, rather than simply affecting individual risk factors (Rose, 2001). A further strength of the ecological analyses presented here is that they may be well-suited to development of policy recommendations that mitigate population level, rather than individual level risks, or maximize population level benefits.

Our focus in this analysis is on geographic variations in multiple social, economic and physical environmental exposures - what has been termed a “riskscape” (Morello-Frosch & Shenassa, 2006) - and their associations with the geographic patterning of mortality outcomes. The methodology used in this analysis, adapted from Su and colleagues (2009), does not lend itself to interpretation of point estimates per unit of change. Rather, its strength lies in the transformation of measures captured with very different units (e.g., percent below poverty, microns per cubic meter for air pollution) into quintiles distributed over a common geographic region. It allows for analysis of the relative distribution of risk across multiple

indicators within a defined geographic area (the DMA) and the independent and joint contributions of those variations in relative risk to variations in mortality.

To our knowledge, this is one of only a handful of papers that have examined multiple characteristics of neighborhoods both individually and collectively, using models that examine their unique contributions to mortality. In doing so, they enable identification of potential mediating pathways and also help to discern potential points of population level intervention that can be useful in identifying priorities for public health intervention and policy change. In the multivariate model presented here (Table 4), indicators of socioeconomic variation (education, income inequality) and exposure to $PM_{2.5}$ retain consistent and statistically significant associations with all-cause mortality, suggesting that interventions to improve educational access and success, reduce income inequality, and interventions to reduce $PM_{2.5}$ have the potential to yield substantial population level reductions in mortality. The findings reported here suggest that, in this DMA analysis, reduced access to education and excess exposure to air pollutants in areas with higher concentrations of NHB residents, substantially attenuate initially observed cross-sectional associations between census tract racial composition and mortality. These findings suggest the importance of further, longitudinal analyses to further examine these associations, and suggest health benefits of efforts to address the maldistribution of educational opportunities and environmental exposures in communities of color.

IMPLICATIONS

In 2001, David Williams and Chiquita Collins (2001) suggested that race-based residential segregation be considered a fundamental cause of health inequities between racial groups, and outlined multiple pathways through which such segregation may contribute to disparities in health outcomes. They concluded that public health interventions focused on reducing racial inequities in health should focus on elimination of race-based residential segregation, or on eliminating the effects of such segregation on access to resources (Williams & Collins, 2001). Our finding that, after accounting for socioeconomic and physical environmental characteristics of neighborhoods, the positive association between proportion NHB residents and mortality is no longer statistically significant joins a body of evidence suggesting that economic divestment and environmental exposures in segregated communities contribute to excess mortality. These results suggest that adverse impacts of race-based residential segregation travel, in part, through impacts of divestment in predominantly black communities for educational and economic opportunities, as well as through the disproportionate exposure of NHB communities to noxious physical environments such as high concentrations of $PM_{2.5}$.

Writing over a decade ago, Frohlich and Potvin (2008) described what they called the “inequality paradox”, in which well-intentioned efforts to improve population health may inadvertently contribute to increases in health disparities as more privileged or well-resourced communities benefit disproportionately compared to structurally marginalized communities. As a result, these population health interventions can inadvertently serve to increase health inequities. One example is the Clean Air Act, passed in the 1970s, which has led to overall reductions in national levels of air pollutants and associated mortality

(Holladay, 2011). However, substantial evidence indicates that, despite these overall reductions in air pollution, communities of color and low income communities remain disproportionately exposed to, and adversely affected by, air pollution and associated health impacts (Ruffin, 2011; Schulz et al., 2018). The inequality paradox, then, is that despite overall reductions in mortality attributable to the Clean Air Act, substantial *inequalities* in exposure and health remain.

To address this paradox, Frohlich and Potvin suggest that interventions must also recognize and address factors that place marginalized populations "at risk of risk." Such efforts may include, for example, investments in public education in order to promote educational and economic equity, and to reduce income inequality while simultaneously working to reduce the inequitable distribution of air pollutants. Additional strategies include addressing race-based housing discrimination as a fundamental driver of racial segregation (Sharkey, 2013), reducing income inequalities (e.g. increases in minimum wage), and promoting more equitable political and economic power to influence land use decisions.

Recognizing that segregation, housing and lending policies in the 20th century were fundamental drivers of today's inequalities in income, education and wealth (Mitchell & Franco, 2018), substantive and concerted efforts to address the contemporary effects of those policies are necessary components of efforts to attain health equity. Examples include community driven community reinvestment strategies that focus on schools, community-based organizations and other anchor institutions located in predominantly Black and Brown areas, which remain more subject to economic inequality (Mitchell and Franco, 2018). It is critical that local development be driven by community insights, values and priorities in order to most effectively address poverty and racism, and avoid unanticipated effects such as gentrification and displacement (National Academies of Sciences Engineering and Medicine, 2017). Specific efforts to address excess exposure to air pollutants in predominantly non-Hispanic Black and Hispanic communities are also essential. Such efforts must be community led (Heaney, Wilson, & Wilson, 2007), and supported with legal, economic and other resources beyond the community, with explicit attention to strategies that avoid, for example, green gentrification and the displacement of current residents as neighborhoods become more desirable to more affluent individuals (Rigolon & Christiansen, 2019). Evidence that countries and states with greater political and economic equity in general generate lower levels of pollution, with more equitable distribution of pollutants, suggest the importance of continued vigilance to protect voting rights and the power to influence land use and other decisions affecting the health and well-being of low income communities and communities of color (Cushing et al., 2015; Wilson, Hutson, & Mujahid, 2008). These and related strategies to promote political, racial and economic equity offer critical pathways toward social and environmental justice and more equitable health outcomes.

White supremacy is a political, economic and cultural system that rests upon the systematic devaluation of Black and Brown lives (Ansley, 1997). We have argued here that it underlies the creation and maintenance of racially segregated communities, economic disinvestment from those communities, and permitting of the emission of excess pollutants affecting those communities. As such, efforts to reduce racial and ethnic health inequities must address

ideologies of white racial superiority imbedded in legal codes, development and permitting processes, policing, and decisions about the geographic distribution of investment in public infrastructure (Rothstein, 2017). These ideologies fundamentally determine the distribution of risk that results in the unnecessary and unjust deaths of thousands of Black and Brown Americans annually (Thakrar, Forrest, Maltenfort, & Forrest, 2018) and must be addressed if we are to attain racial, ethnic and socioeconomic equity in health.

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Highlights:

- Previous studies have demonstrated associations between race-based residential segregation, neighborhood socioeconomic and physical environmental characteristics, and mortality. Few studies have examined independent and joint effects of these multiple neighborhood characteristics and mortality, including potential mediating pathways.
- We find significant associations between census tract race-based residential segregation (operationalized as percent non-Hispanic Black), education (percent with graduate equivalency degree), poverty (percent below poverty), air pollution (ambient PM2.5) and age adjusted all-cause, all race mortality (henceforth all cause mortality) , in the Detroit Metropolitan Area. Income inequality was not significantly associated with race-based residential segregation.
- In multivariate models, education, income inequality and PM2.5 fully attenuated associations between racial composition and all-cause mortality.
- Results are consistent with the hypothesis that race-based residential segregation is associated with heightened all-cause mortality, and that those effects are mediated by education, income inequality, and exposure to air pollution at the census tract level.
- Public health and cross-sector interventions to eliminate race-based residential segregation or to eliminate the maldistribution of educational and economic resources, and environmental exposures, across census tracts could substantially reduce regional inequities in all-cause mortality.

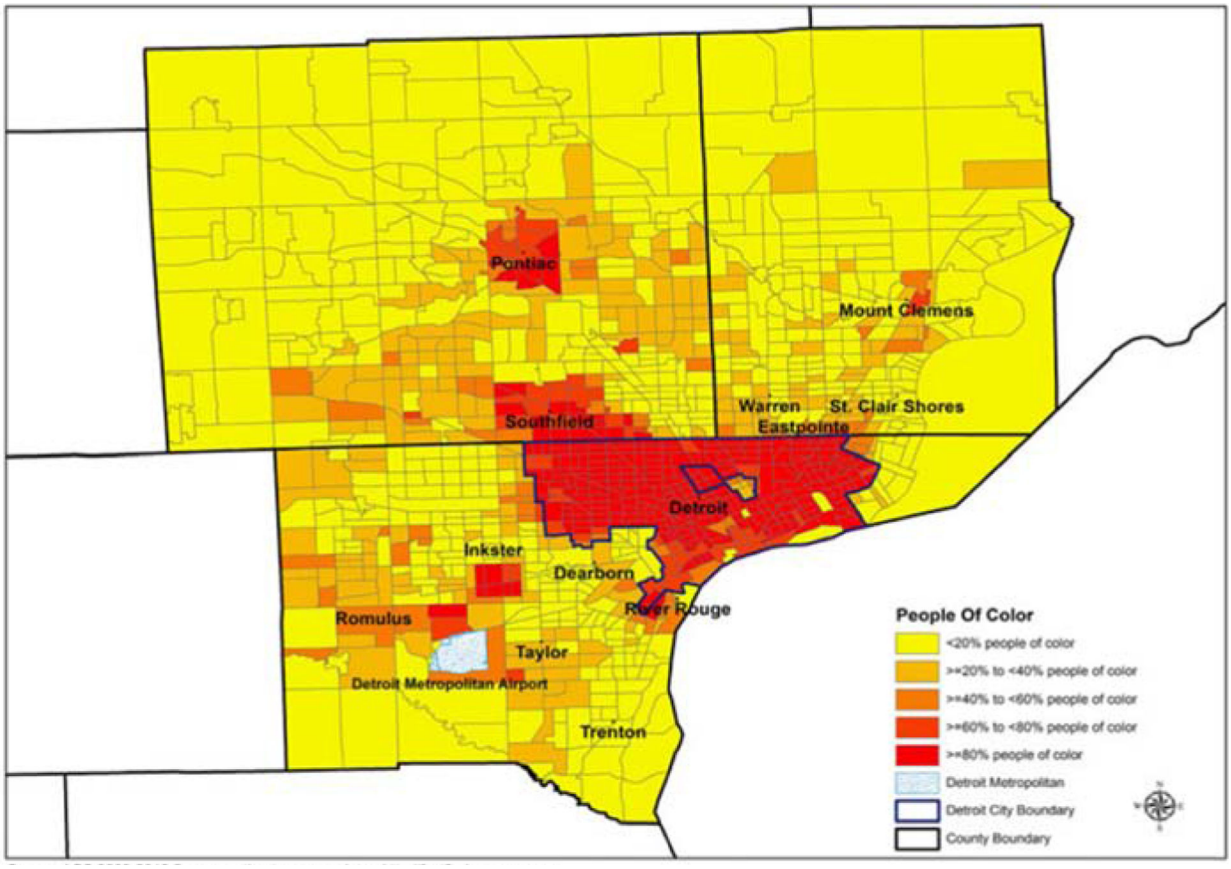

Fig. 1.
Proportion people of color at the Census track level-Detroit Metropolitan Area.

Table 1.

Descriptive Statistics for census tracts reporting mortality (n=1,141) in the Detroit Metropolitan Area (DMA)

Measures	N	%	Mean	StdDev	Min	Max
(continuous)						
All-cause mortality	1141		648.5	235.9	113.0	1418.5
% Non-Hispanic Black (NHB)	1141		29.7	36.9	0.0	100.0
% 25 years and older with <HS education	1141		14.1	10.2	0.0	68.0
% below poverty	1141		17.9	15.4	0.0	69.2
Gini Coefficient	1141		0.4	0.1	0.0	0.7
PM25 concentration	1141		10.2	0.4	9.2	10.8
(categorical)						
%NHB, Quintiles						
1	229	20.1			0.0	1.7
2	229	20.1			1.7	4.8
3	229	20.1			4.8	14.8
4	229	20.1			14.8	83.6
5	225	19.7			83.6	100.0
% 25 years and older with <HS education, Quintiles						
1	229	20.1			0.0	5.6
2	230	20.2			5.6	9.6
3	230	20.2			9.6	14.3
4	230	20.2			14.3	21.9
5	222	19.5			21.9	68.0
% below poverty, Quintiles						
1	230	20.2			0.0	5.1
2	228	20.0			5.1	8.6
3	229	20.1			8.6	16.0
4	230	20.2			16.0	33.6
5	224	19.6			33.6	69.2
Income inequality (GINI) (xl0)						
1	229	20.1			0.0	3.6
2	231	20.3			3.7	3.9
3	228	20.0			4.0	4.3
4	228	20.0			4.3	4.7
5	224	19.6			4.7	7.0
PM2.5 Concentration, Quintiles						
1	231	20.2			9.2	9.9
2	230	20.2			9.9	10.3
3	333	29.2			10.3	10.5
4	124	10.9			10.5	10.5
5	223	19.5			10.5	10.8

Table 2

presents results from analyses examining the first research question: what are the associations between all-cause mortality and racial composition (percent NHB), socioeconomic status (percent of residents aged 25 and older without a high school education, percent below poverty, income inequality), and air pollution ($PM_{2.5}$). In general, all-cause mortality increased for census tracts with greater proportions of NHB residents, greater proportions of residents with lower socioeconomic status, and census tracts with greater exposure to particulate matter. As shown in Model 1, residents of census tracts in the 4th and 5th quintile experienced 40.8 (p=0.055) and 139.9 (p=0.000) more deaths per 100,000 population, compared to those in the first quintile. Mortality rates in the 2nd and 3rd quintiles were not significantly different from those in the lowest quintile of NHB residents.

	Model 1				**Model 2**				**Model 3**				**Model 4**	
Variable (Census tract quintiles, 1 =ref)	**Estimate**	**Stderr**	**95%CI**	**P−value**	**Estimate**	**Stdesrr**	**95%CI**	**P −value**	**Estimate**	**Stderr**	**95%CI**	**P−value**	**Estimate**	**Ssd**
Intercept	608.3	14.4	1580.1636.5)	**0.000**	550.1	13.8	(523.577.1)	**0.00**	592.1	15.2	(562.3622)	**0.000**	591.6	14.4
Percent non −Hispanic Black														
1	ref													
2	*−7.2*	21.5	(−49.5. 35.0)	0.737										
3	28.1	20.7	(−12.4. 68.6)	0.173										
4	40.8	21.3	(−0.9. 82.5)	0.055										
5	139.9	20.9	(99.0,180.8)	0.000										
Percent > age 24 with < HS education														
1					ref									
2					61.7	20.6	(21.4,102.1)	0.003						
3					122.0	20.0	(82.9.161.1)	0.000						
4					150.8	19.7	(112.3, 189.4)	0.000						
5					158.7	22.6	(114.5, 202.9)	0.000						
Percent households below poverty														
1									ref					
2									41.8	21.3	(0. 83. 5)	0.050		
3									63.5	21.2	(22.0, 105.0)	0.003		
4									62.6	20.8	(21.9,103.3)	0.003		
5									114.6	23.5	(68.5,160.8)	**0.000**		
Income Inequality														
1													ref	
2													39.3	19.4
3													S9.2	21.7
4													65.8	20.8

	Model 1				Model 2				Model 3				Model 4	
Variable (Census tract quintiles, 1 =ref)	**Estimate**	**Stderr**	**95%CI**	**P−value**	**Estimate**	**Stdesrr**	**95%CI**	**P −value**	**Estimate**	**Stderr**	**95%CI**	**P−value**	**Estimate**	**Ssd**
5													121.2	23.
PM2.5														
1														
2														
3														
4														
5														

Table 3:

Significance tests of inequality in distribution of neighborhood poverty, education, income inequality, and PM2.5 by neighborhood racial composition in the Detroit Metropolitan Area (census tract n=1144)

		Index	95%CI
Census tract Percent Non-Hispanic Black	Proportion over age 25 and older without high school completion	−0.437	(−0.406,−0.467)***
	Proportion residents living below poverty	−0.580	(−0.536,−0.625)***
	Proportion of residents living in neighborhood with high income inequality (GINI)	−0.410	(−0.382,−0.438)
	Proportion resident living in neighborhoods with high PM25 exposure	−0.485	(−0.451,−0.52)***
	Proportion residents living below poverty	−0.091	(−0.08,−0.101)**

Table 4:

Census tract all cause mortality regressed on percent non-Hispanic Black, percent adult population with less than high school education, income inequality, and fine airborne particulate matter ($PM_{2.5}$)

	Model 1				Model 2				Model 3			
Variable (Census tract quintiles, 1=ref)	**Estimate**	**Stderr**	**95%CI**	**p-value**	**Estimate**	**Stderr**	**95%CI**	**p-value**	**Estimate**	**Stderr**	**95%CI**	**p-value**
Intercept	608.3	14.4	(580.1,636.5)	0.000	527.5	20.1	(488, 567)	<.0001	448.4	20.2	(408.9 , 487.9)	<.0001
Percent non-Hispanic Black												
1	Ref				Ref				Ref			
2	−7.2	21.5	(−49.5, 35.0)	0.737	−16.7	20.8	(−57.5, 24.2)	0.424	−29.4	19.9	(−68.4 , 9.6)	0.140
3	28.1	20.7	(−12.4, 68.6)	0.173	1.7	20.9	(−39.3, 42.6)	0.936	−22.1	20.6	(−62.4 , 18.2)	0.283
4	**40.8**	**21.3**	**(−0.9, 82.5)**	**0.055**	−8.8	23.3	(−54.4, 36.8)	0.706	−36.5	23.5	(−82.5 , 9.6)	0.120
5	**139.9**	**20.9**	**(99.0,180.8)**	**0.000**	**68.6**	**25.7**	**(18.1,119.1)**	**0.008**	32.5	27.5	(−21.3 , 86.3)	0.237
Percent >age 24 with <HS education												
1					Ref				Ref			
2					**64.9**	**20.9**	**(24,105.8)**	**0.002**	**66.4**	**20.3**	**(26.5 , 106.2)**	**0.001**
3					**117.0**	**20.4**	**(77, 157.1)**	**<.0001**	**104.6**	**20.2**	**(65.0, 144.1)**	**<.0001**
4					**122.4**	**22.1**	**(79.2, 165.7)**	**<.0001**	**102.4**	**23.5**	**(56.4 , 148.4)**	**<.0001**
5					**105.7**	**25.8**	**(55.1, 156.3)**	**<.0001**	**87.3**	**27.9**	**(32.6 , 141.9)**	**0.002**
Income Inequality (GINI Coefficient)												
1					Ref				Ref			
2					24.4	18.6	(−12.1,60.9)	0.190	24.5	17.5	**(−9.9 , 58.8)**	0.162
3					38.4	21.1	(−3, 79.8)	0.069	**40.9**	**20.3**	**(1.1 , 80.7)**	**0.044**
4					19.4	22.5	(−24.7,63.5)	0.388	28.5	21.9	(−14.5 , 71.6)	0.193
5					**69.7**	**24.9**	**(21, 118.4)**	**0.005**	**76.7**	**25.1**	**(27.4 , 125.9)**	**0.002**
$PM_{2.5}$												
1									**Ref**			

	Model 1				Model 2				Model 3			
Variable (Census tract quintiles, 1=ref)	**Estimate**	**Stderr**	**95%CI**	**p-value**	**Estimate**	**Stderr**	**95%CI**	**p-value**	**Estimate**	**Stderr**	**95%CI**	**p-value**
2									**144.6**	**20.4**	**(104.5 , 184.7)**	**<.0001**
3									**129.9**	**19.2**	**(92.2 , 167.7)**	**<.0001**
4									**115.2**	**26.2**	**(63.8 , 166.5)**	**<.0001**
5									**131.4**	**25.5**	**(81.5 , 181.4)**	**<.0001**