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Exploration of PM mass, source, and component-related factors that might explain heterogeneity in daily $PM_{2.5}$-mortality associations across the United States

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Abstract

Multi-city epidemiologic studies examining short-term (daily) differences in fine particulate matter ($PM_{2.5}$) provide evidence of substantial spatial heterogeneity in city-specific mortality risk estimates across the United States. Because $PM_{2.5}$ is a mixture of particles, both directly emitted from sources or formed through atmospheric reactions, some of this heterogeneity may be due to regional variations in $PM_{2.5}$ toxicity. Using inverse variance weighted linear regression, we examined change in percent change in mortality in association with 24 “exposure” determinants representing three basic groupings based on potential explanations for differences in PM toxicity – size, source, and composition. Percent changes in mortality for the $PM_{2.5}$-mortality association for 313 core-based statistical areas and their metropolitan divisions over 1999–2005 were used as the outcome. Several determinants were identified as potential contributors to heterogeneity: all mass fraction determinants, vehicle miles traveled (VMT) for diesel total, VMT gas per capita, $PM_{2.5}$ ammonium, $PM_{2.5}$ nitrate, and $PM_{2.5}$ sulfate. In multivariable models, only daily correlation of $PM_{2.5}$ with PM_{10} and long-term average $PM_{2.5}$ mass concentration were retained, explaining approximately 10% of total variability. The results of this analysis contribute to the growing body of literature specifically focusing on assessing the underlying basis of the observed spatial heterogeneity in $PM_{2.5}$-mortality effect estimates, continuing to demonstrate that this heterogeneity is multifactorial and not attributable to a single aspect of PM.

Keywords

Air pollution; particulate matter; mortality; heterogeneity; components

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1 Introduction

Fine particulate matter ($PM_{2.5}$[i]) is a mixture of particles, both directly emitted from sources or formed through atmospheric reactions, with aerodynamic diameters generally of 2.5 micrometers or smaller. Multi-city population-based epidemiologic studies have provided some of the strongest evidence indicating a relationship between short-term (daily) $PM_{2.5}$ exposures and mortality (Baxter et al. 2017; Dai et al. 2014; Di et al. 2017a; Di et al. 2017b; Franklin et al. 2007; Franklin et al. 2008; Krall et al. 2013; Zanobetti and Schwartz 2009), and have heavily contributed to the overarching conclusion that there is a causal relationship between short-term $PM_{2.5}$ exposure and mortality (U.S. EPA. 2009, 2019). While these nationally representative epidemiologic studies provide evidence of positive associations, there is evidence of city-to-city or regional heterogeneity in the magnitude of the $PM_{2.5}$ mortality effect estimates (Baxter et al. 2019; Di et al. 2017b; Dominici et al. 2006; Franklin et al. 2007). The inability to explain the city-to-city or regional heterogeneity in $PM_{2.5}$ mortality effect estimates observed in multi-city studies remains a key uncertainty in the examination of the relationship between short-term $PM_{2.5}$ exposures and mortality.

The observed heterogeneity in $PM_{2.5}$-mortality effect estimates has been hypothesized to be due to differences in source profiles and composition of $PM_{2.5}$ across the United States (U.S.). As a result, numerous studies have examined the relationship between individual $PM_{2.5}$ components and mortality to assess whether the observed heterogeneity can be attributed to some individual $PM_{2.5}$ components being more toxic than others. However, no consistent components have been identified as being more strongly associated with mortality than others. For example, Atkinson et al. (2015) conducted a systematic review of the epidemiologic time-series literature of the relationship between particle components and mortality, finding that sulfate (SO_4^{2-}), nitrate (NO_3^-), elemental carbon (EC), and organic carbon (OC) were positively associated with increases in all-cause, cardiovascular, and respiratory mortality. Additional studies have demonstrated associations with various metal components and mortality, such as aluminum (Franklin et al. 2008), nickel (Franklin et al. 2008; Ito et al. 2011), vanadium (Ito et al. 2011; Lippmann et al. 2013), copper (Lippmann et al. 2013; Ostro et al. 2007), and zinc (Ito et al. 2011; Ostro et al. 2007). The variability in results across these epidemiologic studies indicates that compositional differences in $PM_{2.5}$ do not fully explain the heterogeneity in $PM_{2.5}$ mortality effect estimates across the U.S. This variability is further reflected in an assessment of studies examining $PM_{2.5}$ components and mortality in the 2019 PM Integrated Science Assessment that contributed to the conclusion that, “the evidence does not indicate that any one source or component is consistently more strongly related to health effects than $PM_{2.5}$ mass.” (U.S. EPA. 2019).

More recently, studies have expanded the examination of the observed heterogeneity in $PM_{2.5}$ mortality effect estimates in an attempt to address this question more broadly instead of narrowly focusing on individual $PM_{2.5}$ components. Additional exploration of this uncertainty has led to examinations of whether the heterogeneity in $PM_{2.5}$ mortality

[i]AQS: Air quality system, CBSA: Core Based Statistical Area, CMAQ: Community Multiscale Air Quality modeling system, IQR: inter-quartile range, MD: Metropolitan Division, NCDC: National Climatic Data Center, NEI: Nation Emissions Inventory, PM: particulate matter, VMT Vehicle Miles Traveled

effect estimates can be explained by unique differences in $PM_{2.5}$ component mixtures between cities (Baxter et al. 2013), in the distribution of the population potentially at greatest risk of an air pollutant-related health effect (Levy et al. 2012), and in differences in city-specific exposures to $PM_{2.5}$ (Baxter et al. 2019). While each of these studies provides information to explain some of the observed heterogeneity, together they indicate a complex and multifaceted issue.

Our analyses further examine the issue of spatial heterogeneity in $PM_{2.5}$-mortality effect estimates by focusing specifically on the degree to which PM mass, sources, or composition explain this heterogeneity. Using $PM_{2.5}$-mortality effect estimates from analyses of 312 Core Based Statistical Areas (CBSAs) and Metropolitan Divisions (MD) across the U.S. from 1999–2005, in combination with determinants representing aspects of PM exposure and mass, sources, and components, we explore whether these determinants contribute to the observed spatial heterogeneity in mortality effect estimates using meta-regression techniques.

2 Methods

2.1 Outcome and study population

For this analysis, the study population consists of all residents of 312 core-based statistical areas (CBSA) across the U.S. for which associations (percent change) between daily counts of total non-accidental mortality and daily (24-hour average) ambient concentrations of $PM_{2.5}$ were previously estimated (Figure 1) (Baxter et al. 2019). As detailed in Baxter et al. (2019), associations between daily $PM_{2.5}$ from the EPA's Air Quality System's (AQS) Technology Transfer Network (U.S. EPA. 2020b) and individual level mortality data from the National Center for Health Statistics (http://www.cdc.gov/nchs/about.htm) were estimated at lag 1 for 1999 through 2005 using Poisson time series methods adjusting for time/season (natural spline with 7 degrees of freedom per year), day of week, and natural splines for current temperature, dew point temperature, and individual lagged temperature at lags 1–3 for each CBSA. We selected $PM_{2.5}$ at lag 1 because the largest magnitude effect estimates for $PM_{2.5}$ associated mortality occur within this window (Di et al. 2017a; Franklin et al. 2007; Krall et al. 2013; Ostro et al. 2007; Zanobetti and Schwartz 2009), and to be consistent with previous work (Baxter et al. 2019); in addition, we do not expect spatial determinants to differ between lags 0 and 1. Meteorological data for all U.S. cities were obtained from the U.S. Department of Commerce's National Climatic Data Center (NCDC). The effect estimates for each CBSA are expressed as a percent change in nonaccidental mortality for a 10 $\mu g/m^3$ increase in daily $PM_{2.5}$ 1 day before death and are used here as our outcome measure.

2.2 Spatial determinants

We explored 24 determinants representing three basic groupings based on potential explanations for differences in PM toxicity – aspects of exposure or mass fraction, source, and composition; all determinants, descriptions, and sources are listed in Table 1. These groups have been previously identified as potential contributors to PM toxicity and can affect delivered dose and mode of action (Kelly and Fussell 2012; Lippmann 2012).

Therefore, differences in spatial distributions of these determinants across CBSAs may help explain some of the observed heterogeneity in $PM_{2.5}$-mortality associations.

The first group includes determinants representing aspects of PM related to exposure and mass fractions: long-term average $PM_{2.5}$, correlation between daily $PM_{2.5}$ and daily PM_{10}, etc. This group was chosen in an attempt to elucidate the impacts of overall mass-based exposure metrics, as when the balance of mass is shifted towards larger or smaller particles different associations with health might be observed; though there is extensive evidence indicating health effects attributed to multiple size fractions (U.S. EPA. 2019). Long-term average $PM_{2.5}$ might reflect underlying particle distribution, but may also affect responses to short-term exposures through increased susceptibility (Kunzli et al. 2001; Shi et al. 2016). The ratio of $PM_{2.5}/PM_{10}$ should reflect the proportion of PM_{10} that is comprised of $PM_{2.5}$, while the daily $PM_{2.5}$-PM_{10} correlation may capture days with higher degrees of shared sources between $PM_{2.5}$ and PM_{10}. For these determinants, concentrations of PM were obtained from the EPA's AQS (U.S. EPA. 2020b) within each CBSA and a county-population weighted daily average was created when there were multiple monitors.

The second group consists of determinants representing emissions and sources: $PM_{2.5}$ emissions from wildland fires, diesel and gas emissions vehicle miles traveled, etc. These determinants may represent potential mixtures of chemicals particular to certain sources, some of which may produce biological responses (for example, traffic related) while others may not (for example., soil related) (Kelly and Fussell 2012). This information was sourced from the EPA's National Emissions Inventory (NEI) (U.S. EPA 2015; U.S. EPA. 2020a), and county-level population-weighted averages were created when there were multiple counties within a CBSA.

The final group of determinants consists of specific $PM_{2.5}$ components: ammonium, sulfates, nitrates, elemental carbon, and organic carbons. Component concentrations are known to vary spatially and have been identified and investigated as a source of heterogeneity previously (Kelly and Fussell 2012; U.S. EPA. 2019). For the components group, annual average $PM_{2.5}$ component concentrations were estimated between 1990 and 2010 on a 36×36 km grid using the Community Multiscale Air Quality (CMAQ v 5.0.2) framework (Gan et al. 2015). Estimated concentrations were calibrated against observed concentrations from air quality networks and performed well (correlation coefficients above 0.8 for all component estimates) (Gan et al. 2015). Then, thin-plate smoothing, by means of the R software package "fields" (Nychka et al. 2018), was used to interpolate monthly average concentrations to population centroids of U.S. census tracts, and population-weighted averages were calculated across census tracts and months to obtain annual $PM_{2.5}$ concentrations for each county and year, these were then averaged from 1998–2006 (Peterson et al. 2020).

All exposure determinants are mean-centered and scaled to their respective inter-quartile range (IQR).

2.3 Statistical analysis

Exposure determinants (continuous) were first examined in univariate, inverse variance weighted regression models with percent change in mortality for a 10 μg/m^3 increment in $PM_{2.5}$ as the outcome (i.e., a meta-regression) using the gam package in R (Hastie 2019; R Core Team 2013). Number of CBSAs in each model was allowed to vary with available exposure data. Each beta can be interpreted as the change in $PM_{2.5}$ associated mean percent change in nonaccidental mortality for an IQR increase of the specific determinant, in other words a shift in the percent change in mortality associated with daily $PM_{2.5}$ exposure. Models using natural cubic spline smoothing were also used to check for non-linearity (Fasiolo et al. 2020; Wood 2011), and weighted correlations were also examined. Following bivariable analysis, a final multivariable model was built using a backwards selection approach with the following inclusion criteria: 1) determinants where the univariate models were improvements over the null model, as indicated by a lower BIC; 2) determinants that were roughly linear as determined by spline models; and 3) determinants that were not highly co-linear (correlation coefficient <0.7). For co-linear determinants, the determinant with the higher F-statistic was included (Baxter et al. 2019). In these cases, the selected determinant may be representing the impact of both co-linear determinants. The multivariable model was run iteratively with the least significant determinant dropped at each iteration of the model, until all covariates were significant at the chosen critical level ($p < 0.05$).

3 Results

Distributions of CBSA/metropolitan division-specific health effect estimates and inverse variance weights are displayed on the maps in Figures 1 and 2 (supporting numbers in Supplemental Table S.1); the overall percent change in nonaccidental mortality for a 10 μg/m^3 increase in daily $PM_{2.5}$ 1 day before death was 1.05%, with an IQR of 2.67. More populous areas have larger inverse-variance weights (Figure 2).

Descriptive statistics for spatial determinants are presented in Table 2, with correlations between determinants presented in Supplemental Table S.2. While all included counties (n=312) had values for most $PM_{2.5}$, sources/emissions determinants, and modeled component concentrations, fewer counties had data on PM_{10} concentration, $PM_{2.5}$-PM_{10} daily correlation values, and $PM_{2.5}$ dust construction emissions. Variation across CBSAs was generally low for mass fraction and component determinants, and higher for sources/emissions determinants. Most determinants had moderate to low correlations ($|r| < 0.7$); however, some were highly correlated (e.g., component determinants: ammonium, sulfate, and nitrate) and so were not included together in multi-determinant models.

3.1 Meta-regression results

Within the single determinant meta-regression, several determinants had some level of predictive ability for percent change in nonaccidental mortality for a 10 μg/m^3 increase in daily $PM_{2.5}$ 1 day before death (Table 3). These included all mass fraction determinants, VMT diesel total and VMT gas per capita from sources/emissions determinants, and ammonium, nitrate, and sulfate from PM component determinants. For example, the overall

percent change in mortality is 1.05% (which corresponds to a mortality rate ratio of 1.0105). Therefore, a beta of 0.34, as with the correlation between daily $PM_{2.5}$ and PM_{10} concentrations, can be roughly interpreted as such: a correlation increase from 40% to 58% (IQR increase) would increase the average mortality association from 1.05% to 1.39%. A negative beta would be a decrease in the average mortality association, for example, a 2.72 $\mu g/m^3$ increase in long-term $PM_{2.5}$ average would decrease the average mortality association to 0.75% (beta of −0.30).

The amount of variability in the $PM_{2.5}$-mortality effect estimates explained by any individual determinant was generally low, with the highest adjusted r-squared value being 8.19% for the correlation between daily $PM_{2.5}$ and PM_{10} concentrations. Following that, long-term average $PM_{2.5}$ mass concentration explained 7.86% of the total variation in mortality. All other determinants with some predictive ability explained less than 5% of the total variability in $PM_{2.5}$ associated percent change in mortality, with the $PM_{2.5/10}$ ratio explaining the lowest at 0.69%.

Determinants considered for a multivariable meta-regression model were long-term PM_{10} average, long-term $PM_{2.5}$ average, daily $PM_{2.5}$-PM_{10} correlation, daily $PM_{2.5/10}$ ratio, VMT diesel total, VMT gas per capita, $PM_{2.5}$ ammonium, $PM_{2.5}$ nitrate, and $PM_{2.5}$ sulfate. Of these, $PM_{2.5}$ nitrate was non-linear (see supplemental materials, spline figures). $PM_{2.5}$ ammonium, $PM_{2.5}$ nitrate, and $PM_{2.5}$ sulfate were highly correlated; as $PM_{2.5}$ ammonium had the highest F-statistic it was retained for the multivariable model. The initial multivariable model included long-term PM_{10} average, long-term $PM_{2.5}$ average, $PM_{2.5}$-PM_{10} correlation, $PM_{2.5/10}$ ratio, VMT diesel total, VMT gas per capita, and $PM_{2.5}$ ammonium. Backwards selection was performed until all included determinants were significant at the <0.05 level, leaving a final model that included only two determinants, daily correlation of $PM_{2.5}$ with PM_{10} and long-term average $PM_{2.5}$ mass concentration. Confounding between the two determinants was determined to be meaningful using a 10% change in estimate.

In the ultimate multivariable model, the adjusted percent change in nonaccidental mortality for correlation between daily $PM_{2.5}$ and PM_{10} concentrations was 0.22 (0.06, 0.39), and for long-term average $PM_{2.5}$ mass concentration was −0.17 (−0.31, −0.04). Adjusted r-squared for the final multi-determinant model was 10.38%.

4 Discussion

The goal of this meta-regression analysis was to explore if any of the available determinants related to size, source, or composition explained observed heterogeneity in the association between daily $PM_{2.5}$ exposure and mortality. We identified several individual determinants that accounted for some heterogeneity, though the total variability explained for each was relatively low.

Higher long-term average PM_{10} and $PM_{2.5}$ concentrations were associated with lower daily $PM_{2.5}$-mortality effect estimates, which follows previous work showing greater health

benefit of reductions (steeper relative slope) in $PM_{2.5}$ in those counties achieving attainment compared to those that did not (Corrigan et al. 2018).

Higher correlation between daily $PM_{2.5}$ and PM_{10} was associated with higher daily $PM_{2.5}$-mortality effect estimates. In a study in Spain examining different size fractions, Perez et al. (2009) reported that the coarse fraction ($PM_{10\text{-}2.5}$) was moderately associated with the intramodal fraction ($PM_{2.5–1}$) and almost uncorrelated with PM_1. While these relationships are likely to differ in different locales, it may suggest that when the daily $PM_{2.5}$-PM_{10} correlation is higher $PM_{2.5}$ may be made up of more intramodal particles than when the correlation is low. Sources and conditions that might contribute to high $PM_{2.5}$-PM_{10} correlation include arid locales (windblown dust/dust generation), seasonality, and relative humidity conditions among others (Claiborn et al. 2000; Kegler et al. 2001; Kozákovác et al. 2018). Dosimetric studies have also shown that larger particles do not penetrate and deposit in the lower respiratory tract (U.S. EPA. 2019). The $PM_{2.5/10}$ ratio was also identified as accounting for some heterogeneity, however the amount explained in this case was small (0.7% of total variability).

Emissions related to gas and diesel vehicle miles traveled were both inversely associated with daily $PM_{2.5}$-mortality effect estimates; this might be explained by higher travel being an indicator for poorer exposure capture/measurement error, as the homogeneity across the MSAs may not accurately reflect intra-urban variability in $PM_{2.5}$ concentrations (Dionisio et al. 2014; Dionisio et al. 2016). The $PM_{2.5}$ components of ammonium, nitrate, and sulfate were all positively associated with percent change in $PM_{2.5}$ related mortality. Some of these components have previously been identified as possible contributors to increase the magnitude of the $PM_{2.5}$-mortality relationship (Franklin et al. 2008; Lippmann et al. 2013; U.S. EPA. 2019), and may be associated with specific source profiles for individual cities. However, estimated concentrations of these components were all highly correlated, and they can all be potentially high contributors to $PM_{2.5}$ mass complicating the interpretation of these results.

Of these individual determinants, two were retained in the final multivariable model, correlation between daily $PM_{2.5}$ and PM_{10}, and long-term average $PM_{2.5}$. This may be a function of the level of analysis at the CBSA, where $PM_{2.5}$ mass is more spatially homogenous compared to individual components. It may also be because of measurement in general, as during the time period of this study there was limited data available for components, and emissions may not reflect direct concentrations in the same CBSA due to chemical transport.

Previous studies have investigated potential sources of heterogeneity in the $PM_{2.5}$-mortality association, often focusing on $PM_{2.5}$ components, sources, or related determinants; however, these studies were typically performed in fewer cities than this analysis. Several studies identified season or season-related determinants as a potential source of modification (Dai et al. 2014; Franklin et al. 2008; Lippmann et al. 2013; Zanobetti et al. 2014), suggesting that differences in sources or atmospheric chemistry across time of year are influential on the $PM_{2.5}$-mortality association. Interestingly, a study performed across similar years and in a smaller number of cities did not observe seasonal differences (Krall et al. 2013). Lippmann

et al. (2013) also identified sulfate and carbon monoxide (likely a traffic source indicator), among others, as having consistent associations with mortality in multi-city analyses.

Franklin et al. (2008) also examined the $PM_{2.5}$ component to $PM_{2.5}$ mass proportions as potential modifiers, finding sulfate as well as some metal components to be associated with higher $PM_{2.5}$ related mortality. Some studies have examined regional patterns and city source profiles as a way to tease out potential sources of heterogeneity. Davis et al. (2011) found north-south differences wherein the northern U.S. cities had higher concentrations of sulfate and nitrate but noted that between city heterogeneity remained within regions. Baxter et al. (2013) examined differences in PM components and source profiles using paired cities within regions but did not identify any specific component or sources that could explain heterogeneity in mortality associations between city pairs. Across these studies, heterogeneity in effect estimates seems driven by complex interactions from a variety of determinants and no one component or source was more strongly associated with mortality than others.

This analysis adds to the existing body of literature by including more cities/CBSAs than have previously been included, and by having estimated PM component concentrations for each of these cities. However, potential limitations remain. Temporal variation for cities is likely well captured by central site monitors that are used for daily $PM_{2.5}$ measurement, however these may not capture spatial variability in the sources and components within cities. Similarly, a 36 km^2 model output was used for estimating component concentrations, which involved interpolating to census tracts and then aggregating to MSAs. There is likely uncertainty in estimations of component concentrations as well as in emissions sourced concentrations; low coefficients of variation may indicate a lack of power to detect effects rather than a true absence of effect due to specific components. Relatedly, we were unable to examine metal components which some have identified as potential contributors to PM toxicity, such as nickel or vanadium, as we lacked data on these components. We recognize that the data used in this analysis is older, as those were the data used for the initial mortality effect estimate estimation, and that there have been changes in source contributions to $PM_{2.5}$ over time. For example, the growth in the number of wildfires over time has led to an increase in the proportion of $PM_{2.5}$ emissions from wildfires (U.S. EPA. 2020a). Additionally, over the last 15 years there has been a dramatic change in the contribution of sulfate to overall $PM_{2.5}$ concentrations, specifically in the eastern U.S. which can be attributed to the almost 65% reduction in SO_2 concentrations that have occurred over this time period (U.S. EPA. 2019); from 2003–2005, sulfate accounted for close to 50% of overall $PM_{2.5}$ mass, whereas from 2013–2015 it accounted for about a quarter to a third of mass (U.S. EPA. 2019). In CMAQ analyses, sulfate was the component with the largest total and percent decrease between 1990 and 2010 (−42%) (Peterson et al. 2020). Epidemiologic studies using more recent years of air quality data indicate that this change in the $PM_{2.5}$ mixture does not impact the $PM_{2.5}$-mortality association by demonstrating mortality risk estimates of similar magnitude compared to previous studies. While it is unlikely that this change will contribute to differences in $PM_{2.5}$ effect estimates, it is important to recognize there have been changes in overall $PM_{2.5}$ component concentrations over time. The fairly dramatic change in sulfate contributions to $PM_{2.5}$ mass in the eastern U.S. and the continued relationship between short-term $PM_{2.5}$ exposure and mortality provides evidence supporting

that $PM_{2.5}$ mass remains a good indicator of exposure, and suggests the potential that it is the mixture of $PM_{2.5}$ itself that impacts health rather than an individual component. Numerous studies have shown that many $PM_{2.5}$ components are associated with many health outcomes, including mortality, but no individual component has been shown to be more consistently associated with mortality than $PM_{2.5}$ mass (U.S. EPA. 2019).

5 Conclusions

This study adds to the growing body of evidence indicating that the heterogeneity in $PM_{2.5}$-mortality associations is multifactorial. Whereas the previous hypothesis around the observed heterogeneity was often attributed to variability in the composition of $PM_{2.5}$ across locations, often with a focus on these differences being driven by an individual component, we did not observe such in this analysis. While some components did explain some of the heterogeneity, determinants related to overall $PM_{2.5}$ mass were the strongest predictors. The determinants identified from this analysis can be combined with work done to explore other sources of heterogeneity, such as infiltration determinants and underlying population characteristics, to more fully explain the observed heterogeneity in $PM_{2.5}$ mortality associations across the U. S.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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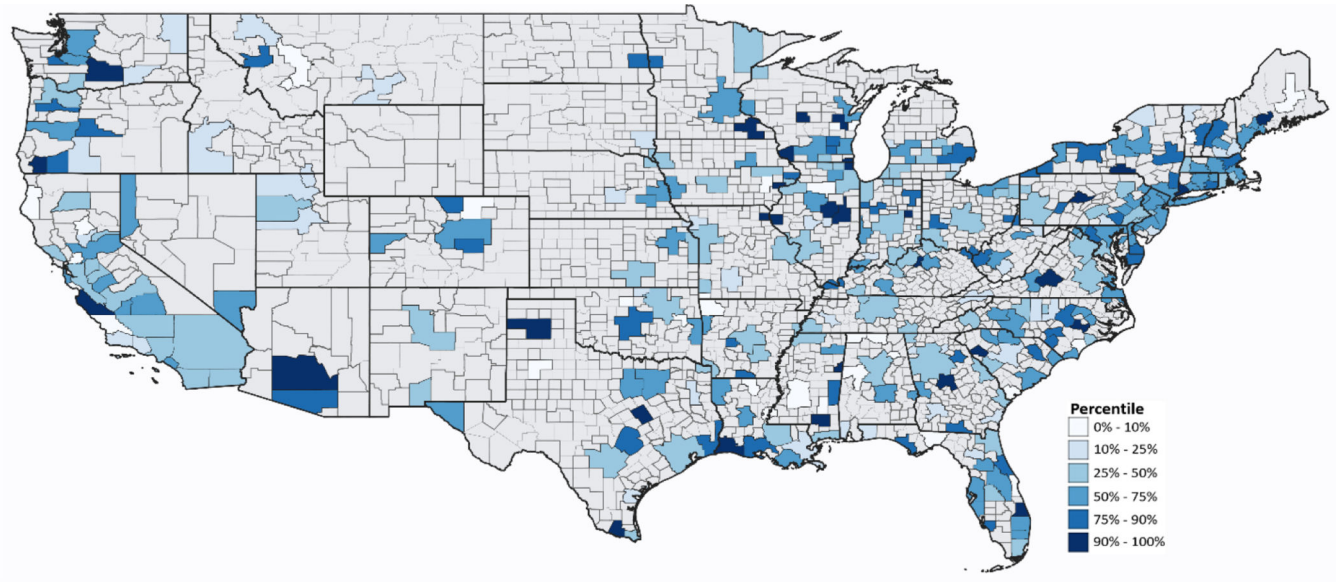

Figure 1:

Area-specific distribution of associations of total non-accidental mortality and fine particulate matter ($PM_{2.5}$) at lag 1: 312 U.S. core-based statistical areas and their metropolitan divisions (Supplemental Table S.1)

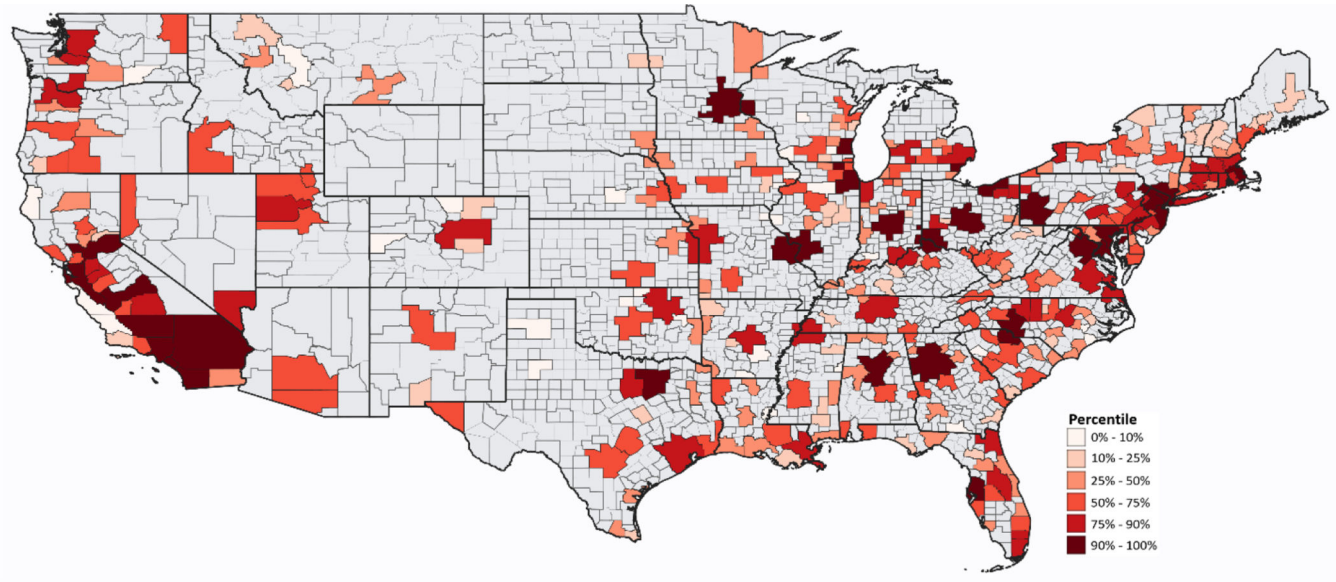

Figure 2:

Area-specific distributions of inverse variance weights for associations of total non-accidental mortality and fine particulate matter ($PM_{2.5}$) at lag 1: 312 U.S. core-based statistical areas and their metropolitan divisions (Supplemental Table S.1)

Table 1:

Descriptions of determinants used to explore spatial heterogeneity in the CBSA-level effect estimates between daily $PM_{2.5}$ and all-cause mortality

Exposure determinant	Group	Description	Source for raw data	Years
Long-term PM_{10} average	Mass fraction	6-year average of daily PM_{10} values at the CBSA level	AQS	1999 – 2005
Long-term $PM_{2.5}$ average	Mass fraction	6-year average of daily PM5 values at the CBSA level	AQS	1999 – 2005
$PM_{2.5}$ - PM_{10} correlation	Mass fraction	Correlation between daily $PM_{2.5}$ and daily PM_{10} at the CBSA level	AQS	1999 – 2005
PM ratio 2.5/10	Mass fraction	Ratio of average $PM_{2.5}$ to PM_{10} concentrations	AQS	1999 – 2005
$PM_{2.5}$ dust construction	Sources/emissions	$PM_{2.5}$ emitted from construction as dust (tons) (NEI technical documentation section 3.7)	NEI	2011
$PM_{2.5}$ dust paved road	Sources/emissions	$PM_{2.5}$ emitted from paved roads as dust (tons) (NEI technical documentation section 3.8)	NEI	2011
$PM_{2.5}$ dust unpaved road	Sources/emissions	$PM_{2.5}$ emitted from unpaved roads as dust (tons) (NEI technical documentation section 3.9)	NEI	2011
$PM_{2.5}$ fires wild	Sources/emissions	$PM_{2.5}$ emitted from wildfires (tons) (NEI technical documentation section 5.1)	NEI	2011
$PM_{2.5}$ fires prescribed	Sources/emissions	$PM_{2.5}$ emitted from prescribed burning (tons) (NEI technical documentation section 5.1)	NEI	2011
$PM_{2.5}$ fires agriculture field burning	Sources/emissions	$PM_{2.5}$ emitted from agricultural field burning (tons) (NEI technical documentation section 5.2)	NEI	2011
$PM_{2.5}$ fires all	Sources/emissions	Summed emissions from fires (tons)	NEI	2011
$PM_{2.5}$ dust all	Sources/emissions	Summed emissions from dust(tons)	NEI	2011
$PM_{2.5}$ dust all and ag crops/ livestock	Sources/emissions	Summed emissions from fires, dust, and agricultural practices in tons	NEI	2011
$PM_{2.5}$ agriculture crops livestock	Sources/emissions	$PM_{2.5}$ emitted from agricultural tilling (tons) (NEI technical documentation section 3.2)	NEI	2011
VMT diesel total	Sources/emissions	Emissions related to vehicle miles traveled, vehicles designed to use diesel fuel, in tons	NEI	2011
VMT diesel per capita	Sources/emissions	Emissions related to vehicle miles traveled, vehicles designed to use diesel fuel, divided by population, in tons	NEI	2011
VMT gas total	Sources/emissions	Emissions related to vehicle miles traveled, vehicles designed to use gasoline fuel, divided by population, in tons	NEI	2011
VMT gas per capita	Sources/emissions	Emissions related to vehicle miles traveled, vehicles designed to use gasoline fuel per capita, in tons	NEI	2011
$PM_{2.5}$ ammonium	Components	Estimated $PM_{2.5}$ ammonium concentration averaged across 9 years	CMAQ	1998 – 2006
$PM_{2.5}$ nitrate	Components	Estimated $PM_{2.5}$ nitrate concentration averaged across 9 years	CMAQ	1998 – 2006
$PM_{2.5}$ sulfate	Components	Estimated $PM_{2.5}$ sulfate concentration averaged across 9 years	CMAQ	1998 – 2006
$PM_{2.5}$ elemental carbon	Components	Estimated $PM_{2.5}$ elemental carbon concentration averaged across 9 years	CMAQ	1998 – 2006
$PM_{2.5}$ organic carbon	Components	Estimated $PM_{2.5}$ organic carbon concentration averaged across 9 years	CMAQ	1998 – 2006

AQS: EPA's Air Quality System (https://www.epa.gov/aqs)

CBSA: Core-based Statistical Area

CMAQ: Community Multiscale Air Quality Modeling System (https://www.epa.gov/cmaq)

NEI: EPA's National Emissions Inventory (https://www.epa.gov/air-emissions-inventories/national-emissions-inventory-nei)

VMT: vehicle miles traveled, these emission estimates are derived by first estimating the VMT and then multiplying by a fixed emission constant.

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Table 2:

Descriptive statistics for spatial determinants

Exposure group	Spatial determinant	Units	N	Mean (SD)	Median (IQR)	Coefficient of variation
Mass fraction	PM_{10} long-term average	$\mu g/m^3$	267	22.42 (6.23)	23.53 (5.91)	0.25
	$PM_{2.5}$ long-term average	$\mu g/m^3$	312	12.59 (3.61)	12.55 (2.73)	0.22
	$PM_{2.5}$-PM_{10} daily correlation		238	0.70 (0.25)	0.67 (0.18)	0.26
	PM ratio 2.5/10		267	0.55 (0.13)	0.56 (0.18)	0.24
Sources/emissions	$PM_{2.5}$ dust construction	tons emitted/year	241	137 (321)	419 (944)	2.25
	$PM_{2.5}$ dust paved road	tons emitted/year	312	301 (392)	513 (597)	1.16
	$PM_{2.5}$ dust unpaved road	tons emitted/year	312	428 (822)	1,065 (2,022)	1.90
	$PM_{2.5}$ fires wild	tons emitted/year	312	32 (248)	712 (3,089)	4.34
	$PM_{2.5}$ fires prescribed	tons emitted/year	312	135 (727)	932 (2,389)	2.56
	$PM_{2.5}$ fires agriculture field burning	tons emitted/year	312	9 (52)	70 (213)	3.06
	$PM_{2.5}$ fires all	tons emitted/year	312	310 (1,527)	1,713 (4,339)	2.53
	$PM_{2.5}$ dust all	tons emitted/year	312	1,062 (1,508)	1,997 (3,090)	1.55
	$PM_{2.5}$ dust all and ag crops/livestock	tons emitted/year	312	1,570 (2,351)	2,699 (3,555)	1.32
	$PM_{2.5}$ agriculture crops livestock	tons emitted/year	312	242 (782)	702 (1,125)	1.60
	VMT diesel total	megatons emitted/year	312	284 (408)	528 (727)	1.38
	VMT diesel per capita	tons emitted/year	312	882 (358)	921 (352)	0.38
	VMT gas total	megatons emitted/year	312	3,150 (5,418)	6,680 (9,801)	1.47
	VMT gas per capita	tons emitted/year	312	9,913 (2,594)	10,191 (1,886)	0.19
Components	$PM_{2.5}$ ammonium	$\mu g/m^3$	312	0.63 (0.40)	0.61 (0.28)	0.47
	$PM_{2.5}$ nitrate	$\mu g/m^3$	312	0.69 (0.64)	0.70 (0.36)	0.52
	$PM_{2.5}$ sulfate	$\mu g/m^3$	312	2.03 (0.93)	1.88 (0.68)	0.36
	$PM_{2.5}$ elemental carbon	$\mu g/m^3$	312	0.29 (0.11)	0.31 (0.10)	0.31
	$PM_{2.5}$ organic carbon	$\mu g/m^3$	312	0.94 (0.30)	0.94 (0.22)	0.23

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Table 3:

Meta-regression single determinant model results

Exposure group	Spatial determinant	beta (95% CI)*	F-statistic	BIC lower than null model?	Adjusted r-squared (%)
Mass fraction	Long-term PM_{10} average	−0.22 (−0.34, −0.09)	11.38	TRUE	3.76
	Long-term $PM_{2.5}$ average	−0.30 (−0.41, −0.19)	27.55	TRUE	7.86
	$PM_{2.5}$ - PM_{10} correlation	0.34 (0.20, 0.48)	22.15	TRUE	8.19
	PM ratio 2.5/10	−0.16 (−0.34, 0.03)	2.85	TRUE	0.69
Sources/emissions	$PM_{2.5}$ dust construction	−0.01 (−0.03, 0.01)	1.04	FALSE	0.01
	$PM_{2.5}$ dust paved road	0.01 (−0.04, 0.06)	0.12	FALSE	−0.28
	$PM_{2.5}$ dust unpaved road	−0.01 (−0.05, 0.03)	0.19	FALSE	−0.26
	$PM_{2.5}$ fires wild	0.00 (−0.01, 0.01)	0.15	FALSE	−0.27
	$PM_{2.5}$ fires prescribed	−0.02 (−0.07, 0.03)	0.71	FALSE	−0.09
	$PM_{2.5}$ fires agriculture field burning	−0.01 (−0.05, 0.03)	0.29	FALSE	−0.23
	$PM_{2.5}$ fires all	−0.01 (−0.06, 0.05)	0.04	FALSE	−0.31
	$PM_{2.5}$ dust all	−0.01 (−0.05, 0.03)	0.38	FALSE	−0.20
	$PM_{2.5}$ dust all and ag crops/livestock	−0.01 (−0.07, 0.04)	0.26	FALSE	−0.24
	$PM_{2.5}$ agriculture crops livestock	0.00 (−0.07, 0.07)	0.00	FALSE	−0.32
	VMT+ diesel total	−0.04 (−0.08, −0.01)	6.28	TRUE	1.67
	VMT diesel per capita	−0.13 (−0.27, 0.01)	3.43	FALSE	0.77
	VMT gas total	−0.03 (−0.05, 0.00)	3.89	FALSE	0.92
	VMT gas per capita	−0.18 (−0.31, −0.04)	6.26	TRUE	1.66
Components	$PM_{2.5}$ ammonium	0.24 (0.11, 0.38)	12.75	TRUE	3.64
	$PM_{2.5}$ nitrate	0.31 (0.11, 0.52)	9.35	TRUE	2.61
	$PM_{2.5}$ sulfate	0.21 (0.08, 0.35)	9.69	TRUE	2.72
	$PM_{2.5}$ elemental carbon	−0.02 (−0.11, 0.08)	0.09	FALSE	−0.29
	$PM_{2.5}$ organic carbon	0.11 (−0.06, 0.29)	1.54	FALSE	0.17

*beta should be interpreted as a shift from the overall daily $PM_{2.5}$-mortality association (percent change in mortality) of 1.05%. For example, for every 5.91 μg/m^3 increase in 6 year average PM_{10} the mean daily $PM_{2.5}$-mortality association decreases by 0.22%.

+vehicle miles traveled