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Warmer is healthier: Effects on mortality rates of changes in average fine particulate matter (PM2.5) concentrations and temperatures in 100 U.S. cities

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ABSTRACT

Recent studies have indicated that reducing particulate pollution would substantially reduce average daily mortality rates, prolonging lives, especially among the elderly (age ⩾ 75). These benefits are projected by statistical models of significant positive associations between levels of fine particulate matter (PM2.5) levels and daily mortality rates. We examine the empirical correspondence between *changes* in average PM2.5 levels and temperatures from 1999 to 2000, and corresponding changes in average daily mortality rates, in each of 100 U.S. cities in the National Mortality and Morbidity Air Pollution Study (NMMAPS) data base, which has extensive PM2.5, temperature, and mortality data for those 2 years. Increases in average daily temperatures appear to significantly reduce average daily mortality rates, as expected from previous research. Unexpectedly, reductions in PM2.5 do not appear to cause any reductions in mortality rates. PM2.5 and mortality rates are both elevated on cold winter days, creating a significant positive statistical relation between their levels, but we find no evidence that reductions in PM2.5 concentrations cause reductions in mortality rates. For all concerned, it is crucial to use causal relations, rather than statistical associations, to project the changes in human health risks due to interventions such as reductions in particulate air pollution.

1. Introduction

In 2011, the United States EPA projected that further reducing levels of fine particulate matter (PM2.5) will significantly extend life expectancy in the United States (EPA, 2011). Similarly, Fann et al. (2012) estimated that "about 80,000 premature mortalities [per year] would be avoided by lowering PM2.5 levels to 5 μg/m^3 nationwide" and that 2005 levels of PM2.5 cause about 130,000 premature mortalities per year among people over age 29, with a simulation-based 95% confidence interval of 51,000 to 200,000. Likewise, a recent, influential, NASA-led study of the computer-predicted benefits of measures to combat global warming concluded that 0.7–4.7 million premature deaths per year would be avoided (and increases in temperatures would be moderated) in the near term by further reducing pollutants such as black carbon emissions (Shindell et al., 2012). Pope et al. (2009) concluded from a regression model of the association between reductions in pollution and changes in life expectancy in 211 county units in the U.S. that "A decrease of 10 μg per cubic meter in the concentration of fine particulate matter was associated with an estimated increase in mean (±SE) life expectancy of 0.61 ± 0.20 year (P = 0.004)." They interpreted the statistical regression coefficient causally, as implying that "A reduction in exposure to ambient fine-particulate air pollution contributed to significant and measurable improvements in life expectancy in the United States," but did not report any results of formal statistical tests (e.g., Bauwens et al., 2006) of this causal interpretation.

Such striking model-projected benefits invite empirical confirmation. The National Mortality and Morbidity Air Pollution Study (NMMAPS, www.ihapss.jhsph.edu/), allows examination of what happened to average daily mortality rates in 100 U.S. cities as PM2.5 levels and temperatures changed between 1999 and 2000, the two years for which most data are available. This paper uses the NMMAPS data (focusing on 1999–2000, but also using the scarcer data from years back to 1987) to compare changes in average daily PM2.5 levels and daily temperatures to corresponding changes in mortality rates.

Past research (Dominici et al., 2007; Mercer, 2003; Healy, 2003) suggests that both PM2.5 and temperature may affect mortality rates. Dominici et al. (2007) examined changes in pollution levels and mortality rates between 1987 and 2000, and found that both decreased. Their paper considered coarse (PM10) as well as fine (PM 2.5) particulate matter, and focused more on PM10 and short-term associations of exposures with mortality at city and

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county levels, using the same data base we did. Their concentration–response modeling consisted of Poisson regression (accounting for over dispersion, i.e., variance larger than the expected value) and hierarchical Bayesian modeling, combining intercounty and within county variability, to identify and quantify concentration–response associations. They conclude that the statistical effect (association) of PM2.5 with mortality is greater than the association of PM10 with mortality. We focus mainly on data from 1999 to 2000 (which are an order of magnitude more plentiful than for earlier years) to examine how quantitatively important are the changes in each factor, PM2.5 and temperature, in explaining corresponding changes in city-specific and month-specific mortality rates.

2. Data and methods

The National Morbidity, Mortality, and Air Pollution Study (NMMAPS) data base, made available on-line by Johns Hopkins at www.ihapss.jhsph.edu/, provides historical daily data from January 1, 1987 through December 31, 2000 on temperature and humidity, pollutant concentration measurements, and mortality counts for 108 U.S. cities, of which 101 are currently populated with at least some PM2.5 data. (PM2.5 data was not collected in all years and days in all cities and often had several-day gaps between data points.) The mortality data include all-cause mortality (excluding accidents) and cause-specific mortality counts, as follows:

- accident – accidental death
- copd – chronic obstructive pulmonary disease
- cvd – cardiovascular deaths
- death – all non-accidental death
- inf - influenza
- pneinf – pneumonia and influenza
- pneu – pneumonia
- resp – respiratory deaths

Dividing these daily mortality counts for the above variables by the population base for each city, year, and age category (from U.S. census data) yields corresponding daily mortality rates by cause, city, year, and age category. (Statistical issues such as heteroscedasticity are dealt with in the subsequent data analysis.) Since most deaths occur among people over 75, we focus on the exposure-mortality association in this age group. For completeness, however, Bayesian model averaging and Granger-Sims causality analyses also consider the two younger age categories in the NMMAPS data set: people under 65 (*agecat* = 1 in NMMAPS) and between 65 and 75 (*agecat* = 2 in NMMAPS). Data are available for over a decade for multiple cities. Hence, they are useful for comparing historical changes in PM2.5 concentrations and corresponding changes in daily mortality rates for different cities.

The NMMAPS data uses a derived variable, *pm25Reconstruct*, to estimate PM2.5 concentration levels. As explained at the iHAPSS web site (http://www.ihapss.jhsph.edu/data/FAQ.html):

> "The median of the trends is stored in a variable with suffix "mtrend". Adding a variable ending in "tmean" with its corresponding "mtrend" variable should get you something resembling the original averaged values. Adding the "tmean" and "mtrend" variables adds the average detrended series with the median of the long term trends from each monitor. It is not an exact reconstruction of any particular series."

Accordingly, we computed *pm25Reconstruct = pm25tmean + pm25mtrend* from the original data, to facilitate cross-city comparisons.

Fig. 1 plots estimated average PM2.5 levels (reconstructed from trend and deviation data for each city, as described in the NMMAPS documentation) and corresponding average daily mortality rates (deaths per million people per day, indicated by the variable "death" in this and subsequent figures) among the elderly (age > 75, who account for most deaths) for 100 U.S. cities, averaged over all 24 months in 1999 and 2000.

The data exhibit significant spread for both PM2.5 and mortality rate, with average estimated PM2.5 levels ranging from under 8 to over 20 μg/m^3 and with average mortality rates per million elderly people per day (the "death" variable shown on the vertical axis) ranging from under 160 in Honolulu to almost 340 in Anchorage (an outlier), and to over 220 in many cities. (To avoid crowding, only selected city names are displayed, showing the most-polluted and least-polluted locations.) Fig. 2 confirms that, although PM2.5 levels and death rates are strongly autocorrelated, there is also substantial variation in their city-specific values from year to year,

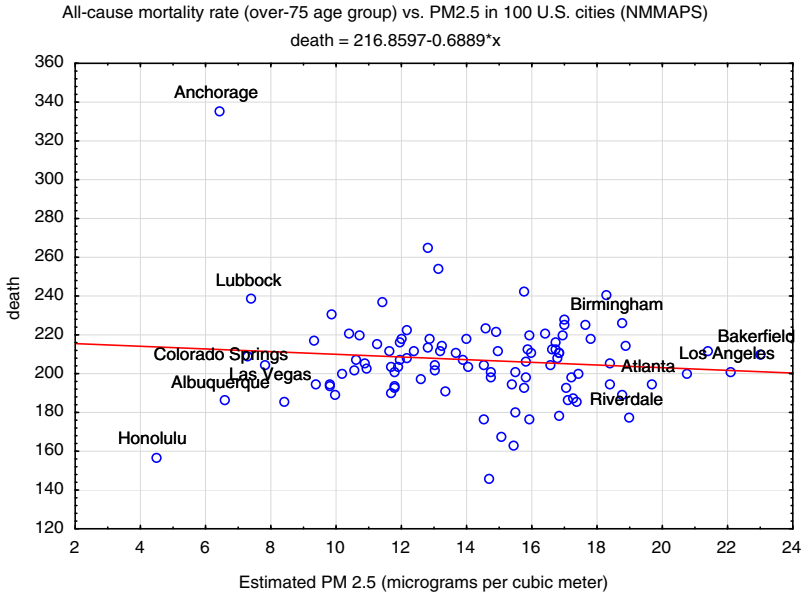

Fig. 1. Average PM2.5 concentrations and daily mortality rates (deaths per million people per day) in 100 U.S. cities, averaged over all 24 months in 1999 and 2000.

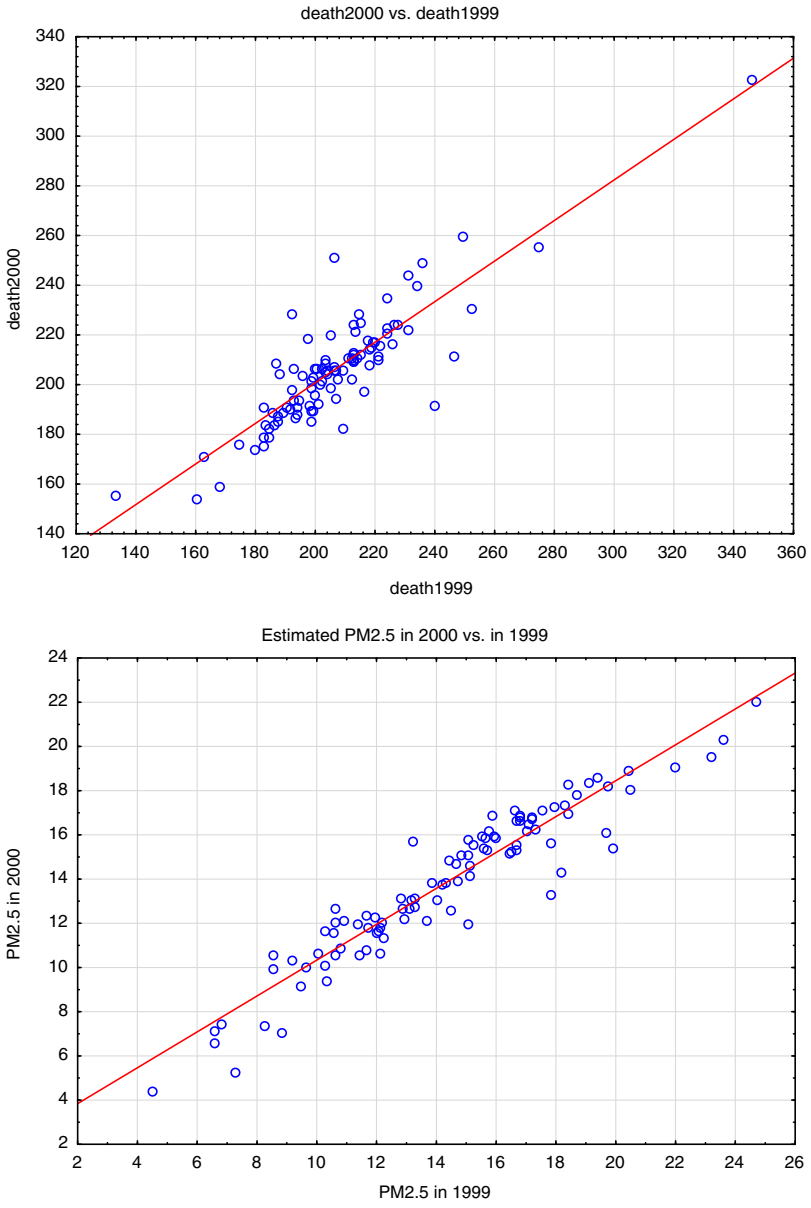

Fig. 2. Mortality rates (deaths per million people per day) for elderly (> 75) and PM2.5 levels are autocorrelated, but vary substantially for individual cities between 1999 and 2000.

allowing an exploration of how changes in city-specific PM2.5 correspond to changes in city-specific mortality rates. (We focus throughout on the elderly (over 75) population, for whom mortality rates are highest; for completeness, results for younger age groups are included in the section on Granger causality testing.)

A policy maker untrained in statistics might wonder why Fig. 1 does not show a positive association between PM2.5 levels and mortality rates. (The regression slope is non-significantly negative.) A statistician might guess that unknown ecological biases obscure a genuine positive relation at the individual level, e.g., if cities with higher PM2.5 levels happen to be healthier in other ways than cities with lower PM2.5 levels. But the question deserves a more definitive answer. NMMAPS is one of two data sets relied on in recent projections of large mortality-reduction benefits from further reductions in PM2.5 (Fann et al., 2012). If ecological biases obscure positive relations, what becomes important is how actions that reduce PM2.5 might affect off-setting factors that eliminate positive aggregate association between average PM2.5 and average mortality rates. In short, the absence of an apparent positive association in Fig. 1 presents a puzzle worth understanding.

To better understand not just association, but also potential causation, in this data set, we first examine how mortality rates changed between 1999 and 2000, as monthly average PM2.5 levels and daily temperatures changed, for the same months and cities. To avoid possible model selection biases, which have previously been discussed for PM2.5-health effects data (Roberts and Martin, 2010; Koop and Tole, 2004), we use nonparametric measures of association (Spearman's rank correlations). This focus on changes in explanatory and dependent variables from year to year, for the same cities and months, implicitly controls for effects of cross-city differences in demographics, locations, and other slowly-changing variables in each city. As in other longitudinal panel design studies, each city-month pair serves as its own control (Croissant and Millo, 2008). Each city's average mortality rate for elderly people, for each month in 2000, is compared to its own past value (lagged by a year), using changes in average monthly PM2.5 levels and

temperature variables as potential explanatory variables for changes in mortality rate. Over a year, PM2.5 and temperature may change significantly (Fig. 2), but demographics and other potential confounders relatively little; thus, the contributions of changes in the quickly-varying variables of PM2.5 and temperature to changes in mortality rates can be seen more clearly than in ecological cross-sectional comparisons such as Fig. 1.

Next, we disaggregate the data to the daily level, and test for potential causation between city-specific daily time series of temperatures and PM2.5 concentrations and mortality rates, using the Granger test for possible causal relations between time series variables. This is available in the R library as the *granger.test* procedure (http://rss.acs.unt.edu/Rdoc/library/MSBVAR/html/granger.test.html). Its documentation explains that *granger.test*:

> "Estimates all possible bivariate Granger causality tests for m variables. Bivariate Granger causality tests for two variables *X* and *Y* evaluate whether the past values of *X* are useful for predicting *Y* once *Y*'s history has been modeled. The null hypothesis is that the past *p* values of *X* do not help in predicting the value of *Y*. The test is implemented by regressing *Y* on p past values of *Y* and *p* past values of *X*. An *F*-test is then used to determine whether the coefficients of the past values of *X* are jointly zero. This produces a matrix with $m*(m-1)$ rows that are all of the possible bivariate Granger causal relations. The results include *F*-statistics and *p*-values for each test. Tests are estimated using single equation OLS models."

We will apply this test to each city in the NMMAPS data set, for all years (1987–2000), for both daily PM2.5 and daily temperature variables (lagged from 1 to 7 days) as candidate causes of daily mortality rates, for all time intervals with at least 50 consecutive days of data (to support time series analysis with days as the time step). (There are 190 such sequences for PM2.5 and mortality rates.)

3. Analyses and results

3.1. Qualitative directional associations

A first exploratory step is to assess how well the directions of changes in PM2.5 and temperatures explain directions of changes in corresponding mortality rates. To do so, we pair data for each month and city in 1999 with data for the same month and city in 2000. With complete data, this would give 100 cities × 12 months = 1200 pairs. Because some cities do not have complete data for all months in both years, the actual number of city-month pairs with data is 1105. Table 1 shows the fraction of these city-month pairs in which the city- and month-specific average daily mortality rate among the elderly increased between 1999 and 2000, broken down by whether average temperature (specifically, minimum daily temperature, averaged over the month) and daily PM2.5 level (also averaged over the month) increased. (The average minimum and average maximum temperatures are closely correlated, and either can be used without changing the conclusions.)

Qualitatively, increases in PM2.5 in Table 1 are not associated with increases in mortality rates. However, increases in temperature are associated with reduced mortality rates (frequency of increased mortality rates is reduced from 0.54 to 0.47 among city-month pairs with no increase in PM2.5, and from 0.49 to 0.45 among city-month pairs with some increase in PM2.5). Quantitatively, Fisher's exact test reveals no significant association between increases in PM2.5 and increases in mortality rates ($p = 0.23$ in a two-sided test and 0.12 in a one-sided test), and a borderline significant association between increases in temperature and reductions in mortality rates ($p = 0.06$ in a two-sided test and 0.03 in a one-sided test).

Table 1
Breakdown of mortality rate increases by changes in PM2.5 and temperature from 1999 to 2000.

tmin (minimum daily temperature) increased?	PM2.5 increased?	Fraction of city-month pairs with increased elderly mortality rate from 1999 to 2000	*N*
No	No	0.54	303
No	Yes	0.49	255
Yes	No	0.47	284
Yes	Yes	0.45	263

Table 2
Ordinal associations between variables in 1999 and 2000. (Variable abbreviations are described in text. All deltas refer to changes between 1999 and 2000.)

Variable	Spearman rank order correlations Marked correlations are significant at $p < .05000$		
	death1999	death2000	deltaMortalityRate
PM25_1999	0.03	0.07	0.05
PM25_2000	0.09	0.11	0.02
delta PM2.5	0.08	0.05	−0.06
tmax1999	−0.48	−0.41	0.13
tmin1999	−0.46	−0.41	0.12
tmax2000	−0.46	−0.42	0.09
tmin2000	−0.45	−0.42	0.08
delta tmax	−0.00	−0.11	−0.13
delta tmin	−0.01	−0.13	−0.14

Ordinal correlations are for all 100 cities and for all 12 months in1999–2000.

3.2. Ordinal correlations

Quantitative comparison of changes in PM2.5 and temperature and corresponding changes in mortality rates gives statistically significant results consistent with the directional findings in Table 1. Table 2 summarizes Spearman's rank correlation coefficients for different pairs of variables, using "delta" to refer to changes between 1999 and 2000.

In Table 2, the rows list potential causes (PM2.5 levels, temperatures, or changes in these variables) and columns list potential effects (the city- and month-specific mortality rates among people over 75 in 1999 ("*death1999*") and 2000 ("*death2000*"), and changes in these mortality rates between 1999 and 2000 ("*deltaMortalityRate*")). All associations with Spearman correlations greater than 0.05 or less than −0.01 in this table are statistically significant at $p < 0.05$.The following significant associations are conspicuous in Table 2:

1. *Warmer temperatures are associated with lower death rates:* For a given city and month, warmer days (indicated by higher average values of daily *tmin* and *tmax*) tend to have lower mortality rates. This is consistent with previous research on temperature and mortality rates: although extreme heat waves in summer can also be deadly, by far the quantitatively most important effect of temperature on mortality is increased mortality rates during cold winter days (e.g., Healy, 2003; Mercer, 2003). Warmer temperatures in 1999 are significantly associated with increases in death rates between 1999 and 2000 (Spearman's rank correlation coefficients are 0.13 and 0.12 for the association between *deltaMortalityRate* and maximum and minimum daily temperatures in 1999, respectively). This is consistent with regression to the mean, if cities with unusually warm weather in a month in 1999 (and hence fewer deaths) are likely to experience colder weather (and hence more deaths) for the same month in 2000.
2. *Increases in temperature are associated with reductions in mortality rates:* Spearman's rank correlation coefficients are −0.13 and −0.14 for the associations between changes in mortality rates and changes in maximum and minimum daily temperatures,

Table 3
Ordinal associations for all cities and years, 1987–2000.

Variable	Spearman rank order correlations MD pairwise deleted Marked correlations are significant at $p < .05$				
	Year	death	tmax	tmin	PM2.5
Year	1.00	−0.01	−0.00	0.07	−0.23
death	−0.01	1.00	−0.18	0.02	−0.09
tmax	−0.00	−0.18	1.00	0.66	0.10
tmin	0.07	0.02	0.66	1.00	−0.02
PM2.5	−0.23	−0.09	0.10	−0.02	1.00

Ordinal correlations are for all 100 cities and for all years, 1987–2000.

respectively. (Although increases in temperatures appear to be beneficial on average in this data set, this does not deny that heat waves in the summer can pose excess mortality risks.)

3. *PM2.5 levels in 2000 are positively associated with mortality rates in 2000* (with a Spearman's rank correlation coefficient = 0.11): This association is not apparent in Fig. 1 because it does not display monthly data. In most cities, the months with the highest PM2.5 levels (typically, December–February) also have the highest mortality rates, although mortality rates are not associated with PM2.5 levels within months. This creates a non-causal association between PM2.5 and mortality rates whenever months or seasons, rather than years, are used as time steps. (PM2.5 levels in 2000 are also significantly associated with mortality rates in 1999. Since the city- and month-specific PM2.5 levels in 1999 and 2000 are autocorrelated, with a Spearman rank correlation of 0.70 (not shown), it is not surprising that PM2.5 in 2000 is correlated with mortality rates in 1999, given that it is correlated with mortality rates in 2000.) In univariate linear regression modeling, city- and month-specific levels of PM2.5 in year 2000 are significant predictors of corresponding city-and month-specific mortality rates in 2000; but in multivariate analysis, this significant association disappears ($p = 0.46$) after conditioning on contemporaneous temperatures (*tmin*). Thus, temperature is a confounder (colder months have statistically significant higher city-specific PM2.5 levels and mortality rates), and this confounding fully explains the (unconditional) significant positive association between PM2.5 levels and mortality rates.

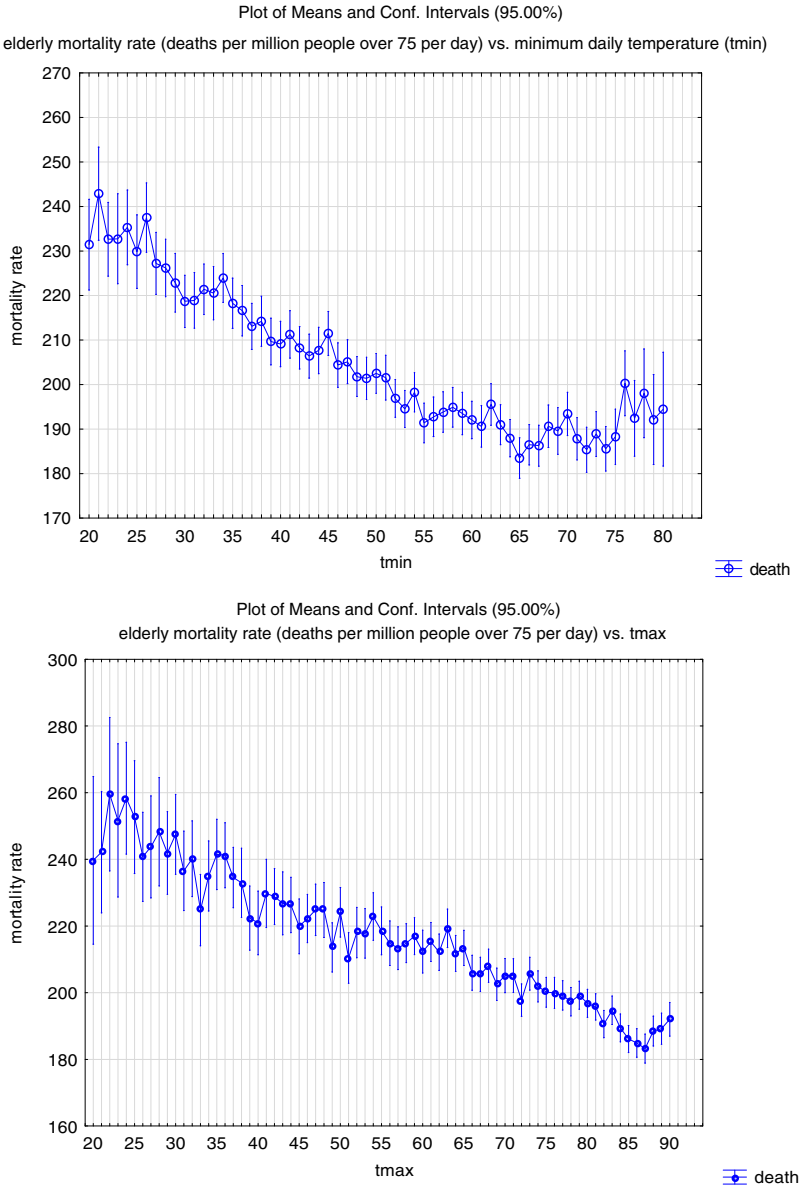

Fig. 3. Warmer daily temperatures are associated with lower daily mortality rates. (Data averaged over all cities and days with available data, 1987–2000.)

4. *Changes in PM2.5 level between 1999 and 2000 are significantly negatively associated with changes in mortality rates*, i.e., for the same month and city, increases in PM2.5 are weakly but significantly associated with decreases in mortality rates (Spearman's rank correlation of −0.06): This unexpected finding shows that the association between *levels* of PM2.5 and mortality rates can be positive, even though the association between *changes* in levels of PM2.5 and changes in mortality rates is negative. It is the latter – effects of changes, rather than associations between levels – that should be of greatest interest for risk managers contemplating interventions that seek to change PM2.5 levels.

3.3. Ordinal associations over longer time horizons

Although the data collected in NMMAPS prior to 1999 is too sparse to allow the panel data design in Table 2 to be applied to the years from 1987 to 1998 (since most days and months in most cities have missing data prior to 1999), Table 3 shows the ordinal correlations between pairs of variables, treating each city in each year as one observation (with an average value in that city and year for each variable). (In Table 3, missing data (MD) pairwise deletion means that any data point with missing value is deleted and it is not replaced by data imputation methods. The NMMAPS data base includes data from 1987 to 2000, however the time series for 1999 and 2000 are the most complete. These data are used for most of our analyses, unless otherwise noted) All correlations greater than 0.15 or less than −0.15 in this table are statistically significant at $p < 0.05$. Thus, PM2.5 is negatively associated with year (average pollution levels declined from 1987–2000 in this sample of cities). Mortality rate (*death*) is negatively associated with *tmax* on this decadal time scale (warmer years and cities have lower mortality rates), and maximum and minimum daily temperatures are strongly correlated with each other across cities and years, as

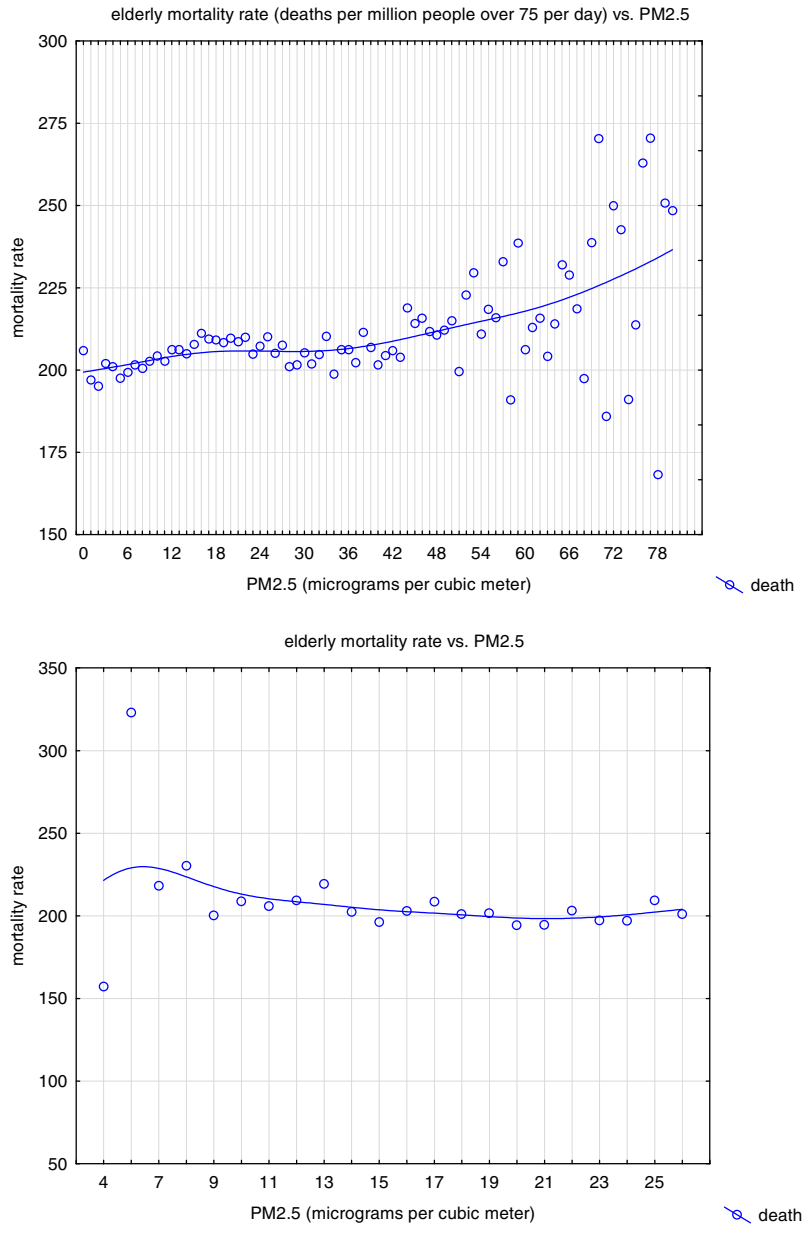

Fig. 4. Mortality rate vs. PM2.5, based on monthly averages (left) and annual averages (right). If it is included, month of year confounds the PM2.5-mortality association. Scatter plot on left is for all cities, months, and years; scatter plot on right is for all cities and years. Smooth curves are fit via nonparametric regression (distance-weighted least squares).

might be expected. There are no other significant associations between variables. (As in the analysis of 1999–2000 data, however, changing the time step from years to months induces a significant positive association between PM2.5 and mortality rate, indicating that, within each year, high PM2.5 months (mainly December–February) are also high-mortality rate months.)

Fig. 3 shows the full relation between daily temperatures (*tmin* and *tmax*) and corresponding daily mortality rates, averaged over all cities and days for which data were collected in the period 1987–2000. This highly statistically significant relation between warmer temperatures and lower mortality rates is probably causal, since Table 2 shows that daily mortality rates fall when temperatures rise.

Fig. 4 shows two versions of the analogous relation between PM2.5 and mortality rate. On the left, a time step of months is used; as expected, this induces a positive association between PM2.5 and mortality rates, as both are highest in cold months. On the right, the time step is years, and there is no evidence of such a positive association, consistent with Tables 2 and 3. (The PM2.5 scales differ in these two plots because monthly averages have more variability, and hence a wider range, than yearly averages. Heteroscedasticity increases at higher PM2.5 values on the monthly time scale, but at lower PM2.5 values on the yearly time scale. Missing data patterns also differ, suggesting some of the complexities that make accurate quantitative modeling of such data challenging, and sensitive to specific modeling decisions (Koop and Tole, 2004).)

3.4. Granger causality tests

Comparing changes over time in hypothesized causes and their hypothesized effects, as in Table 2, provides one strategy for evaluating whether a hypothesized causal relation is supported by data. A different strategy is to determine whether the future of the hypothesized effect variable can be *predicted* better from the history of the hypothesized cause variable (as well as the effect's own history) than from the history of the hypothesized effect variable alone. This motivates the Granger test for potential causality between two time series variables. The R implementation of this test, applied to the NMMAPS data, regresses present and future mortality rates against past ones, and also against past ones together with past values of PM2.5, to determine whether PM2.5 helps to predict mortality rates. We applied this *granger.test* procedure to city-specific daily data on PM2.5 and mortality rates (for all sequences of length at least 50 days, to permit reliable estimation of autoregression and cross-regression coefficients).

Table 4 summarizes the results of Granger testing for PM2.5 as a possible cause of mortality (denoted by "PM2.5 → death"), and also for each of the following cause-specific mortality rates, which are included in the NMMAPS data set (discussed earlier).

The results are presented for each of three age categories, rather than just for the elderly (>75) age group. Table 5 presents analogous results of Granger tests for daily temperatures (specifically, *tmin*) as a cause of mortality rates. Since the Granger tests use daily data, rather than monthly or yearly time steps, they test for potential short-run causal relations. The Granger test procedure applied an *F*-test to determine whether including present and lagged values of PM2.5 as predictors significantly improves ability to predict daily mortality rates, compared to just using lagged values of mortality rates as predictors. Table 4 entries show fractions of daily time series sequences that support the hypothesis that increases in PM2.5 helps to predict increases in daily mortality rates. Values less than 0.05 in Table 4 do not support this hypothesis, but could plausibly be due to chance (at the conventional significance level of $p = 0.05$). Table 5 provides similar information for temperature as a potential predictor of daily mortality rates. Past literature has shown that PM2.5 values lagged by 0–2 days are significantly associated with daily mortality rates (e.g., Franklin et al., 2007). Although this is consistent with our findings in Table 2 for associations between levels of variables, Table 4 indicates that this association does not help to improve predictions (the Granger causality criterion). (To make sure than no lagged effects were missed, the results in Tables 4 and 5 allowed all values lagged by 1–7 days as potential predictors, well beyond the usual 0–2 days range for significant associations.) On the other hand, Table 5 shows that warmer temperature is a clear Granger-cause of reduced daily mortality rates in this data set.

The Granger tests provide little or no evidence of a positive causal relation between PM2.5 and any mortality rate (despite possible confounding by temperature, which might have been expected to have created the appearance of such a relationship), but they show a clear Granger-causal relation between temperature and mortality rates. Specifically, 24 tests of PM2.5 as a possible cause of each of eight types of death (including accidental) in each of three age groups, conducted for all data sequences (i.e., time series) with at least 50 consecutive days in the NMMAPS data set, yielded a fraction of "significant" results well under 5%, consistent with the null hypothesis of no effect (at $p < 5\%$). (Borderline significance was achieved for accidental deaths among 65–75 year olds and for COPD and all-cause mortality in the over-75 age group, but multiple testing without adjustment of the significance levels and without control for potential confounding by temperature weakens the significance of these borderline cases.) By contrast, the null hypothesis of no effect is rejected at $p = 5\%$ in all 24 of 24 cases for *tmin*: the fraction of data sequences in which

Table 4
Granger tests show no significant causal association between PM2.5 and mortality rates in daily time series. Table entries show fractions of daily time series sequences (typically, consistent with the chance level of 5%) that support the hypothesis that increases in PM2.5 helps to predict increases in daily mortality rates (with p-value < 0.05 and positive coefficients for PM2.5-mortality association).

Relation	Age category			
	<65	65–75	>75	Avg.
PM2.5 → accident	0.044	0.058	0.048	0.050
PM2.5 → copd	0.029	0.038	0.050	0.039
PM2.5 → cvd	0.009	0.044	0.041	0.031
PM2.5 → death	0.023	0.036	0.051	0.037
PM2.5 → inf	0.002	0.022	0.014	0.013
PM2.5 → pneinf	0.052	0.041	0.036	0.043
PM2.5 → pneu	0.055	0.039	0.038	0.044
PM2.5 → resp	0.037	0.039	0.045	0.040

Fractions of sequences with p-value $\leqslant 0.05$ and positive correlation.

Table 5
Granger tests strongly reject the null hypotheses of no significant causal association between temperature (*tmin*) and mortality rates in daily time series. Table entries show fractions of daily time series sequences (typically, far greater than the chance level of 5%) that support the hypothesis that increases in temperature helps to predict reductions in daily mortality rates (with p-value < 0.05 and positive coefficients for PM2.5-mortality association).

Relation	Age category			
	<65	65–75	>75	Avg.
tmin → accident	0.17	0.09	0.15	0.14
tmin → copd	0.25	0.34	0.49	0.36
tmin → cvd	0.33	0.41	0.69	0.48
tmin → death	0.40	0.45	0.77	0.54
tmin → inf	0.14	0.14	0.37	0.22
tmin → pneinf	0.37	0.29	0.63	0.43
tmin → pneu	0.34	0.27	0.62	0.41
tmin → resp	0.40	0.42	0.66	0.49

Fractions of sequences with p-value $\leqslant 0.05$ and most correlations are negative.

Granger-causation appears to be present is greater than $p = 5\%$ in every case.

Most of our analyses have used non-parametric methods to avoid model uncertainties, since treatment (or ignoring) of model uncertainty can greatly affect conclusions about exposure-mortality associations (NRC, 2002; Koop and Tole, 2004; Roberts and Martin, 2010). The *granger.test* procedure in this section, however, relies on parametric models and methods (linear autoregression and cross-regression models fit to time series via ordinary least squares). Hence, it is vulnerable to the usual array of questions about model selection and specification. No tests or transformations were attempted to correct for possible nonlinearities, heteroscedasticity, measurement error, interactions of temperatures over time, or other issues: we simply applied the off-the-shelf *granger.test* procedure (except that, for Table 4, we modified the R code to report the direction, as well as the significance, of associations). Thus, the results in Tables 4 and 5 are subject to model uncertainties, and more sophisticated models could be developed within the Granger testing framework. Despite these potential limitations, the results provide some additional confirmatory evidence, using a different analytic strategy from the panel data comparisons, supporting the conclusion that temperature (unlike PM2.5) has a clear causal impact on health outcomes.

3.5. Quantitative estimation of health effects

Two questions of great practical interest to public health risk managers and policy makers are the following (Fann et al., 2012; Shindell et al., 2012): What would happen to human mortality rates if PM2.5 levels were reduced? And, what would happen to human mortality rates if changes in pollutants (or other interventions) changed average temperatures? To help address these questions empirically, Figs. 5 and 6 plot observed changes in city- and month-specific PM2.5 and temperature (*tmin*) levels between 1999 and 2000, for the 100 NMMAPS cities, against corresponding changes in mortality rates.

Consistent with the parametric modeling results from the Granger test and the nonparametric results in Table 2, the empirical results in Figs. 5 and 6 suggest that changes in temperature can have a substantial causal impact on mortality rates. Since the base rate is about 200 mortalities per million people over 75 per day (Fig. 4), an increase or reduction of about 20 deaths per day (towards the left and right ends of the curve in Fig. 4) corresponds to roughly a 10% increase or decrease in mortality rate, respectively. A 7° (Fahrenheit) increase in average *tmin* value might reduce mortality rates by about 10%. By contrast, consistent with the Granger test results, increases in PM2.5 levels within the range of values measured in this study (annual averages of about 4 to about 25 $\mu g/m^3$ (Fig. 4) and changes of up to 8 $\mu g/m^3$) have no apparent effect on increasing mortality rates.

Taken at face value, these results suggest a simple explanation for the puzzle raised by the cross-sectional data in Fig. 1: Cities with lower levels of PM2.5 do not have lower daily mortality rates (when both variables are averaged over a full year) because current levels of PM2.5 have no detectable causal impact on increasing mortality rates. However, warmer cities (and months and days) tend to have lower mortality rates than colder ones (Fig. 3), and this association is very likely to be causal (Table 5). None of this denies that significant positive statistical associations may exist between average *levels* of PM2.5 and average mortality rates in statistical analyses that use time steps of less than a year (e.g., Fig. 4, left side). But, in this data set, such positive statistical associations do not necessarily correctly predict how *changes* in PM2.5 levels affect changes in daily mortality rates. For this latter, causal, relation, the null hypothesis of no positive relation cannot be rejected (Table 3 and Fig. 6).

4. Discussion: Implications for health risk analysis of PM2.5 reductions and temperature changes

Recent predictions that "about 80,000 premature mortalities would be avoided by lowering PM2.5 levels to 5 $\mu g/m^3$ nationwide" (Fann et al., 2012) and that 0.7–4.7 million premature deaths per year can be avoided by measures that reduce exposures to PM2.5 and other pollutants (Shindell et al., 2012) clearly contrast with the finding that reductions in PM2.5 levels have no detectable impacts on reducing mortality rates. Both cannot be correct (at least in the same data set). This section discusses possible reasons for the different conclusions, and possible lessons for risk analysis of air pollution health effects.

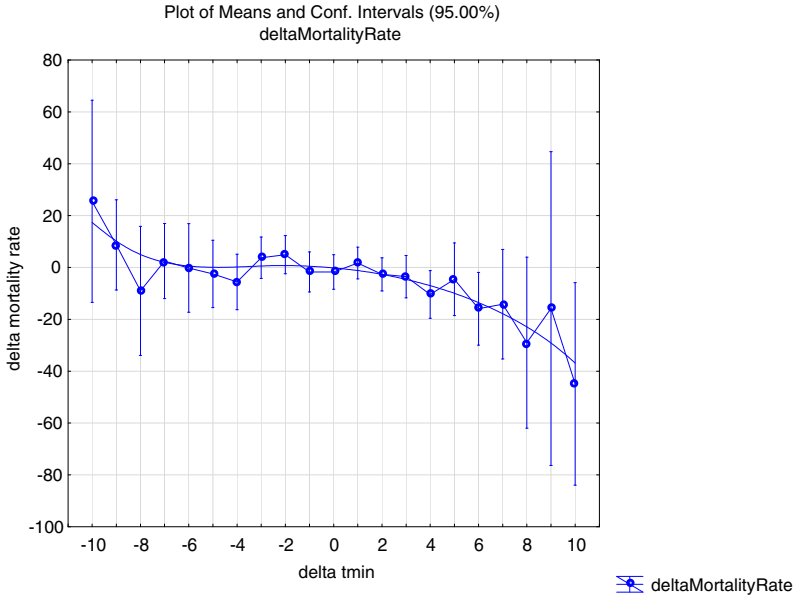

Fig. 5. Empirical relation between changes in temperature (delta *tmin*) and changes in elderly mortality rate (delta mortality rate) between 1999 and 2000 (for the same cities and months).

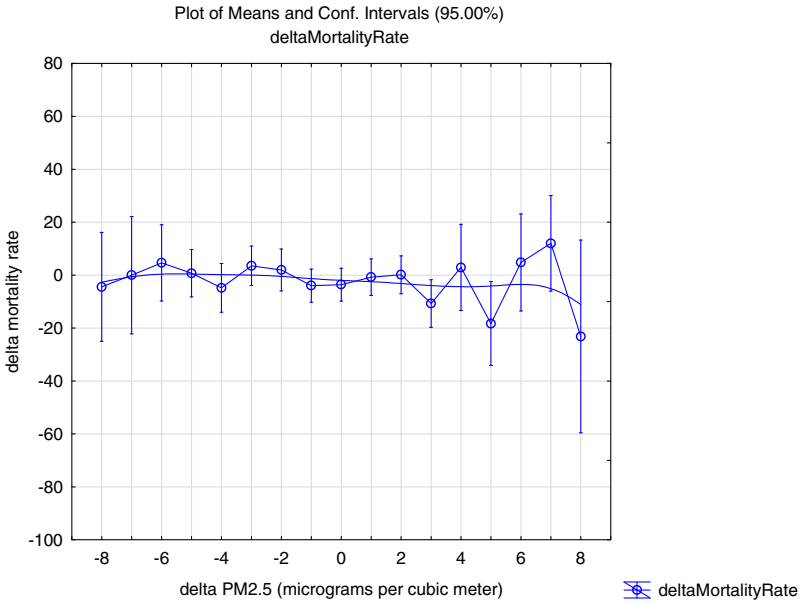

Fig. 6. Empirical relation between changes in PM2.5 (delta PM2.5) and corresponding changes in elderly mortality rate (delta mortality rate) between 1999 and 2000 (for the same cities and months).

4.1. Implications for risk assessment

That statistical associations between PM2.5 levels and mortality rates can be significantly positive, even while the statistical associations between changes in PM2.5 and corresponding changes in mortality rates are significantly negative (Table 2), highlights the fact that *statistical relations are, in general, quite distinct from causal relations.* This is well understood in econometrics, where it motivates the distinction between reduced-form and structural equations models (Cox, 2009). It implies that statistical models of associations between PM2.5 levels and mortality rates are not in general sufficient for predicting the directions or magnitudes of causal impacts on human health of changes in pollutant levels, as attempted in Fann et al. (2012) and Shindell et al. (2012). To correctly predict causal impacts, one needs either a causal model, or a valid design and analysis strategy (such as panel data analysis, a quasi-experimental design, or a time series intervention analysis) that is explicitly appropriate for causal inference (*ibid*). The main lesson of Table 2 for risk assessors may simply be the reminder that causal predictions should not be made from non-causal associations and models.

Fig. 4 suggests how statistical modeling might unintentionally create a spurious positive association between PM2.5 and mortality rates, due to residual confounding by temperature (or season). To control for potential confounding by season, temperature, humidity, and so forth, it is now standard practice among papers such as those cited in the introduction to use splines (or other statistical models) to estimate and subtract out the statistical effect of these variables on mortality rates. But such statistical models have limited flexibility, and hence constrained ability to model the correct relation between temperature (or other quickly-varying explanatory variables) and mortality rates. The degree of adjustment that modelers make for trends, temperature, and seasonality (e.g., choice of degrees of freedom and knot locations) can affect apparent associations, with different statistical models producing different conclusions (e.g., Dominici et al., 2003). Including more degrees of freedom can eliminate statistically significant-seeming PM2.5-mortality associations induced by less flexible models (e.g., Ostro et al., 2006). Thus, risk assessments that use moderately flexible statistical models to represent effects of seasonality may thereby introduce a partly controlled confounder that creates a spurious PM2.5-mortality association. (To avoid this problem, our analyses conditioned on individual months, without any further smoothing or modeling to control for seasonality; or averaged over entire years as time steps, thus avoiding potential confounding by incompletely modeled seasonality.) Fig. 7, from Franklin et al. (2007), illustrates the types of results that have resulted from previous analyses of associations between PM2.5 and mortality rates in different communities, typically using regression-based methods. Similar to the left side of our Fig. 4, such studies usually – but by no means always – show a positive concentration–response association. It would be interesting to reexamine how, if at all, these associations would change if non-parametric comparisons similar to those we have performed were used instead.

A final possible lesson for risk assessment is that quantifying human health benefits from measures that reduce particulate pollutant and slow global warming should, logically, require quantifying changes in mortality rates caused by reduced temperatures, as well as any changes caused by less polluted air. Although associations between mortality rates and seasonal temperatures have previously been discussed (Mercer, 2003; Healy, 2003), to our knowledge, this is the first exploration of the relation between changes in temperatures (and in PM2.5 levels) and corresponding changes in mortality rates over a year-long interval for multiple cities. Analyses that consider only the estimated effects from reduced pollution, but not the effects from changes in temperature, do not address all of the health impacts of proposed interventions. If only changes in temperature, but not in PM2.5, have causal impacts on changing health (with warmer temperatures causing reductions in mortality rates), then this is a serious omission.

4.2. Implications for risk perceptions

Air pollution reduction measures induce a positive emotional response on many grounds (e.g., clean air is attractive), and, via the affect heuristic, this makes it easy to perceive and believe in other benefits (e.g., that such measures will have beneficial health effects) and to overlook or dismiss possible health harms (Slovic et al., 2002). The truth may be more nuanced. In this study, it appears that reducing current levels of PM2.5, however attractive

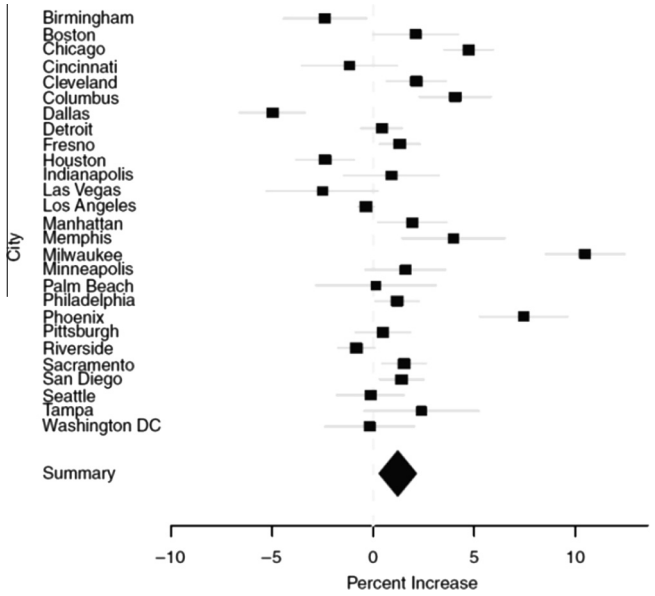

Fig. 7. Previous estimates of the% increase in all-cause mortality per 10 mg/m3 increase in previous day's PM2.5 concentration. (Grey lines are estimated 95% confidence intervals; diamond is overall mean-of-the-means point estimate.) Source: Franklin et al., 2007.

and appealing it may be on other grounds, should not be expected to save lives or increase life expectancy. If it produces cooler temperatures (Shindell et al., 2012), then this might increase mortality rates in some locations, depending on details such as how many additional cold days per year will occur (not quantifiable based on the NMMAPS data) (Mercer 2003; Healy, 2003).

4.3. Implications for risk and uncertainty communication

Recent articles and reports on estimated or predicted human health benefits from PM2.5 reductions have communicated results to policy makers as central estimates surrounded by ranges of plausible values (e.g., a positive interval between upper and lower confidence limits). For example, Fann et al. (2012) refer to 130,000 premature mortalities per year among people over age 29, with a 95% confidence interval of 51,000 to 200,000. Similarly, EPA (2011) adopts a Weibull (continuous, non-negative) uncertainty distribution and uses it to present subjective uncertainty intervals around point estimates. Shindell et al. (2012) report a positive range of 0.7–4.7 million premature deaths per year that would be avoided by the measures they advocate.

Such presentations of risk impacts and uncertainties, as point estimates with surrounding uncertainty intervals, do not quantify the probability that there is no causal impact. Yet, this may be the most important uncertainty for risk management policy making. For example, seven experts who provided much of the scientific input for EPA's quantification of PM2.5-mortality associations assessed a probability for a causal relation between PM2.5 and mortality at 7 $\mu g/m^3$ that ranged from 35% to 99%, with a mean of 81% (EPA, 2006). Non-EPA experts might assess lower values. Thus, presenting a positive uncertainty interval, with no probability for the possibility of a zero causal relation, does not adequately communicate this crucial uncertainty.

4.4. Implications for risk management and risk research

Taking seriously the possibilities that warmer average temperatures decrease elderly mortality rates, but that reducing current levels of PM2.5 might not reduce mortality rates (unless it increases temperatures), invites new questions for health effects researchers, risk analysts, and policy makers. How large must the probability be that further reductions in PM2.5 will not reduce human health risks, before current recommendations advocating such additional reductions become unattractive? How does the answer compare to the current best estimates of this probability? If increases in average temperature from one year to the next reduce mortality rates, does this effect last, or do people become acclimated to warmer average temperatures, and return to previous mortality rates? How do changes in PM2.5 affect the variability of temperatures (e.g., the frequency of severely cold days in the midst of generally mild winters, which may be especially hazardous (Healy, 2003))? Does the weak but significant negative association between changes in PM2.5 and changes in mortality rates in Table 2 represent a real biological effect (e.g., hormesis), or does it have some other explanation?

Addressing such questions will provide a clearer understanding of the probable long-run and short-run impacts on mortality of changes in the statistics of daily temperatures, which may in turn be affected by changes in PM2.5 (Shindell et al., 2012). Such an understanding of probable consequences of proposed interventions is needed for sound risk management decision-making.

5. Conclusions

This paper has examined the changes in average daily mortality rates that occurred in 100 U.S. cities following naturally occurring changes in temperature and PM2.5 levels (on corresponding daily, monthly, and yearly time scales). There is clear evidence of a significant negative Granger-causal relation between temperature and mortality rates, with warmer days corresponding to lower average mortality rates. However, surprisingly, there is no evidence of a positive causal relation between PM2.5 concentration and mortality rates. These findings are inconsistent with recent model-based projections of substantial reductions in mortality rates caused by measures to reduce PM2.5 levels and temperatures.

The question of whether changes in PM2.5 levels actually cause changes in mortality rates should be examined using study designs and analysis methods appropriate for valid causal analysis and inference. The results may differ from projections based on models of statistical associations, which have so far played a central role in air pollution health effects modeling and policy discussions. Improved causal analysis is an essential foundation for improved prediction of the probable public health consequences of policy interventions that affect PM2.5 and temperatures. Risk analyses that help to decisively resolve current scientific uncertainties about causation will be of great value in informing future risk management policy deliberations.

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