Race, Gender, and Social Status as Modifiers of the Effects of PM_{10} on Mortality

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Interest has recently been focused on which populations are most at risk of premature mortality induced by air pollution. This coincides with greater concern about environmental justice. We analyzed total mortality in the four largest US cities with daily measurements of particulate matter less than 10 μm (PM_{10}) and combined the results to determine whether race, sex, and education are potential modifiers of the effects of PM_{10} on mortality. We computed daily counts of deaths stratified by sex, race, and education in each city and investigated their associations with PM_{10} in a Poisson regression model. We combined the results by using inverse variance weighted averages. We found evidence of effect modification by sex, with the slope in female deaths one third larger than in male deaths, whereas for social factors and race we found only weak evidence of effect modification. In general, the effect modification appeared modest compared with other reports of substantial effect modification by medical conditions.

Many studies have assessed the association between daily deaths or hospital admissions and air pollution, both in Europe and in the United States.[1–22] The most widely reported associations are with particulate air pollution. Although these associations are now well documented, it is still unclear as to who are the more sensitive groups. The National Academy of Science, in its Research Priorities for Airborne Particulate Matter, pointed out that it is important to understand the characteristics of individuals that put them at increased risk of adverse events due to particulate air pollution.[23]

Recent attention has been focused on environmental justice. This refers to the question of whether environmental hazards are inequitably distributed by class and race.[24–26] Such differences may be due to differences in exposure, differences in susceptibility, or both. There is a need for more research to define the problem, understand the causes, and identify solutions.[25–31] Many studies have analyzed differences in health by race and gender, and some emphasis has been given to the importance of social class as a predictor of health.[32–35] Recent studies have shown that differential mortality exist by both race and socioeconomic status.[36–40] Socioeconomic inequalities in mortality and morbidity have been found also in Europe[41] and in Brazil.[42] Whether these differences lead to different responses to airborne particles is relatively unexamined. In the time series analysis of short-term effect of air pollution on health, very few studies addressed

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effect modification to date; those that did looked at the difference mainly by age groups.[43–52]

We decided to examine whether race, sex, and education are potential modifiers of the effects of airborne particles on total mortality. In this study, we analyzed the four largest cities across the United States with daily measurements of PM_{10} during the period for which we had mortality data. This multicity analysis, which enabled us to average out city-specific variability, allowed us to investigate the potential effect modification by education, race, and sex.

Data and Methods

Data

Most US cities measured PM_{10} for 1 day in 6, but a number of locations had monitors on a daily schedule. The selection of the cities for this study was based on the number of daily measurements and on the population size. The four largest cities with daily PM_{10} monitoring during our study period (1986–1993) were Chicago, Illinois; Detroit, Michigan; Minneapolis–St. Paul, Minnesota; and Pittsburgh, Pennsylvania. For Minneapolis and St. Paul, we combined the two metropolitan counties associated with these two neighboring cities and treated them as one city.

The 1990 population of the metropolitan counties for these cities varied between 1,336,449 inhabitants in Pittsburgh, Pennsylvania, and 5,105,067 in Chicago, Illinois. In all of the four cities, we had a total of 782,502 deaths over the study period.

Daily meteorological measurements, such as mean temperature, dew point, and barometric pressure, were obtained for each county from the nearest airport weather station. These data were extracted from the EarthInfo Surface Airways CD (EarthInfo CD NCDC Surface Airways, EarthInfo Inc, Boulder, CO).

Daily concentrations of PM_{10} were obtained from the US Environmental Protection Agency's Aerometric Information Retrieval System monitoring network. We excluded days when air pollution exceeded the ambient air quality standard of 150 $\mu g/m^3$ PM_{10} to study the association at common concentrations.

Daily deaths were extracted from the National Center for Health Statistics mortality tapes for the years 1986 through 1993. Deaths due to external causes (ICD-9: 800–999) were excluded. Starting in 1990, the number of years of education was coded on death certificate tapes. Education is considered to be one of the most important social factors in health inasmuch as it relates to income, class, degree of control in job, etc.

We computed separate daily counts of deaths stratified by sex, race, and education to test the hypothesis that persons in each of these groups had higher risks of PM_{10}. Education was stratified on the basis of whether or not the decedent had some postsecondary education.

Methods

In each city, the associations between daily deaths and PM_{10} were investigated with a generalized additive robust Poisson regression model.[53] In the generalized additive model, the outcome is assumed to depend on a sum of nonparametric smooth functions for each variable, which model the potential nonlinear dependence of daily deaths on weather and season. The model is of the form: $\log[E(Y_t)] = \alpha_0 + S_i(X_i) + \ldots + S_p(X_p)$, where $E(Y_t)$ is the expected value of the daily count of deaths Y_t, and S_i are the smooth functions of the covariates X_i. The locally weighted running-line smoother, loess,[54] was chosen to estimate the smooth function.

We fit a model in each city to allow for city-specific differences. The variables included in each model were season, weather variables (temperature, dew point, and barometric pressure), and day of the week, but because weather and season vary across the cities, the smoothing parameter for each variable was optimized separately in each location. To control for weather variables and day of the week, we chose the smoothing parameter that minimized the Akaike's Information Criteria.[55] To model seasonality, we chose the smoothing parameter that minimized the sum of the autocorrelation of the residuals while removing seasonal patterns.

In some cities, autoregressive terms[56] were added in the model to eliminate the remaining serial correlation from the residuals. The mean of PM_{10} on the day of death and the previous day was used as exposure variable and was treated linearly.

We subdivided total mortality counts by the effect modifier (sex, race, and education) and estimated our regressions on those subgroups. We considered effect modification to be indicated when the combined estimate of PM_{10} in one group was outside of the 95% confidence interval (CI) of the effect estimate in persons of the opposite strata.

In the second stage of the analysis, we used the inverse variance weighted averages to combine results across cities. If we assume that the effect of PM_{10} is: $\beta_j^* \sim N(\mu, V)$, where β_j^* is the estimated effect in each city, we can then estimate μ and its variance from the results in the four cities by computing an inverse variance weighted average.

Results

Table 1 shows the 25th, 50th, and 75th percentiles of each of the environmental variables used in the analysis. The values for particles represent the distribution of the mean of the concurrent and previous day of PM_{10}. The distribution of the environmental variables is similar in the four cities.

Chicago, Detroit, and Minneapolis–St.Paul PM_{10} had a moderate number of missing values for PM_{10}, which were mainly concentrated in

the beginning of the 2922-day study period. Pittsburgh had essentially complete data.

Table 2 shows the mean daily counts of total mortality by sex, race, and education. Table 3 shows the populations in each city by the same strata, and the death rates per 10,000 persons. The distribution of the deaths by sex is almost even; there are fewer daily deaths in persons with higher education. The city with the highest percentage of death in people with higher education is Minneapolis–St. Paul. Detroit had the lowest percentage. Conversely, the city with the lowest percentage (4%) of blacks is Minneapolis–St. Paul, and the city with the highest is Detroit. A similar distribution is shown in Table 3 in terms of percentage of population.

Table 4 shows the results of the analysis, expressed as the percentage increase in daily deaths for a 10 $\mu g/m^3$ increase in PM_{10}. The results across city are relatively homogeneous.

The strongest evidence of effect modification was by sex. The percentage increase in female deaths is 1.08 (95% CI, 0.85–1.31) compared with 0.74% (95% CI, 0.53–0.96) for male deaths. For each sex, the combined estimate is outside of the 95% CI for the opposite sex.

The differences were similar, but somewhat smaller, when stratified by race or education and were not statistically significant. However, the effect of PM_{10} on total mortality tended to be higher for whites compared with blacks, and for persons with low education level compared with persons with higher education level.

Discussion

Regardless of significance level, the differences in the effects of PM_{10} on daily deaths among the various strata were modest. For sex, there was significant effect modification with the slope in female deaths one third larger than in male deaths. The reasons for these differences are unclear and deserve further investigation. Deposition of particles in the lung has been shown to vary by gender,[57,58] with greater lung deposition fractions of 1-μM particles in all regions for women. Most of the PM_{10} in these cities is composed of combustion particles, so deposition may represent part of the explanation. Many of the toxic components of cigarette smoke are in the particle phase, and some studies have shown that women are more sensitive than men to the effect of smoking on the risk of mortality, chronic obstructive pulmonary disease (COPD), asthma, and myocardial infarction.[59–61] Other studies have shown that the effects of smoking on lung function may be greater in women than in men.[62–64] To the extent that particles from tobacco act through similar mechanisms as outdoor combustion particles (eg, inflammation), these results may be relevant for our findings that the effect of PM_{10} is higher in women than in men. In patients with COPD, Sunyer and coworkers[65] reported a larger effect of particles on the risk of mortality for women than for men.

Persons with less than a high school education had about a 25% higher PM_{10} slope than persons with at least some college. The first explanation for this result that must be considered is chance. Indeed the difference was not statistically significant, making chance a likely candidate. However, persons with COPD have been shown to be at higher risk

TABLE 1
Percentile Values (25th, 50th, 75th) for the Environmental Variables in the Four Cities: 1986–1993

City	Temperature (°F)	Dew Point	Barometric Pressure	PM_{10} 0–1 ($\mu g/m^3$)	No. Missing
Chicago	35	27	29.2	25	773
	51	39	29.3	33	
	67	55	29.4	44	
Detroit	35	64	29.2	22	479
	50	27	29.3	33	
	67	39	29.4	47	
Minneapolis	30	22	29.0	18	434
	47	34	29.1	25	
	66	53	29.2	35	
Pittsburgh	37	28	28.6	22	2
	53	41	28.8	31	
	68	56	28.9	46	

TABLE 2
Mean ± Standard Deviation of the Daily Deaths Stratified by Sex, Education, and Race in the Four Cities

City	White	Black	Male	Female	Education <12 y	Education >12 y
Chicago	96.1 ± 12.2	36.1 ± 6.5	69.0 ± 9.3	64.4 ± 9.3	100.9 ± 12.0	25.8 ± 5.5
Detroit	35.5 ± 6.8	24.0 ± 5.2	31.5 ± 5.9	28.3 ± 5.8	46.8 ± 7.5	8.9 ± 3.1
Minneapolis	30.5 ± 5.9	1.2 ± 1.1	15.4 ± 4.0	16.9 ± 4.3	21.7 ± 5.0	9.2 ± 3.0
Pittsburgh	37.7 ± 6.9	4.6 ± 2.2	20.9 ± 4.8	21.5 ± 5.1	32.0 ± 6.2	6.8 ± 2.7

of particles-induced mortality.[65,66] COPD is primarily due to smoking, and smoking is more common in persons without a college education in the United States. Hence, further investigation into this question may be fruitful.

The finding of larger effects on whites than on blacks contradicts our original hypothesis that blacks, because of their poorer average health status, might be at higher risk. Again, chance seems to be the most likely explanation for the higher slope in whites. Nevertheless, these findings certainly are inconsistent with a larger effect on blacks, and given the large minority populations in Chicago and Detroit, this is not likely due to a lack of power. One possible explanation is that particulate air pollution risk is not generally higher in persons with poorer health, but rather is restricted to specific conditions not overrepresented in minority groups. Blacks have a lower rate of smoking prevalence than whites, and in the third National Health and Nutrition Examination Series, their rate of chronic bronchitis was lower than for whites (7.4% vs 4.9%).

In contrast to these findings of modest evidence for interactions versus social factors, there is evidence in the literature for more substantial effect modification by medical conditions, in addition to the higher risk in subjects with COPD reported earlier. For example, Schwartz reported a higher risk of cardiovascular deaths associated with airborne particles in subjects with respiratory infections.[52]

Airborne particles also have been associated with systemic changes, including decreased heart rate variability,[67–69] increased peripheral neutrophils,[70–72] increased plasma viscosity,[73] increased heart rate[74,75] and increased blood pressure,[76] which could be potential sources of specific effect modifications.

In conclusion, we found that the association between mortality and PM_{10} was higher in female subjects than in male subjects, but we observed only weak evidence of a modest effect modification due to social factors, race, and gender. Although many studies pointed out differences in health due to social factors, race, or gender, the association between mortality and particulate air pollution could depend more on preexisting medical conditions than on social factors, or the effects of race and class may be apparent only in persons with specific medical conditions. Further work will be needed to evaluate this question.

TABLE 3
Percentage of Total Population by Sex, Race, and Education and Annual Death Rates (per 100,000 Persons)

	Chicago	Detroit	Minneapolis	Pittsburgh
Total population*	5.11	2.11	1.52	1.34
% Black	0.26	0.40	0.05	0.11
% Female	0.52	0.53	0.52	0.53
% Education >12 years†	0.30	0.25	0.39	0.30
Death rates				
White	109	107	82	118
Black	100	103	53	112
Male	103	115	77	122
Female	89	93	78	111

* In millions.
† Persons aged ≥25 years.

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TABLE 4
Effect Modification by Sex, Race, and Education; Percentage Increase in Deaths (95% CI) for 10 ($\mu g/m^3$) of PM_{10} Mean Lags 0–1*

City	White	Black	Male	Female	Education <12 y	Education >12 y
Chicago	0.91 (0.59–1.23)	0.80 (0.31–1.28)	0.89 (0.53–1.25)	0.83 (0.45–1.21)	0.75 (0.37–1.13)	0.87 (0.13–1.61)
Detroit	0.92 (0.56–1.29)	0.64 (0.20–1.08)	0.46 (0.09–0.84)	1.26 (0.84–1.68)	1.03 (0.54–1.52)	0.35 (−0.75–1.46)
Minneapolis–St. Paul	1.35 (0.78–1.92)	1.17 (−1.70–4.13)	0.68 (−0.11–1.48)	1.91 (1.16–2.67)	1.65 (0.70–2.61)	0.69 (−0.77–2.17)
Pittsburgh	0.99 (0.63–1.35)	1.20 (0.22–2.18)	0.95 (0.48–1.42)	0.93 (0.46–1.40)	0.90 (0.29–1.50)	0.72 (−0.57–2.02)
Combined	0.98 (0.80–1.17)	0.76 (0.46–1.07)	0.74 (0.53–0.96)	1.08 (0.85–1.31)	0.92 (0.66–1.18)	0.71 (0.19–1.23)

* CI, confidence interval; PM_{10}, particulate matter less than 10 μm.

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