Air Conditioning and Source-Specific Particles as Modifiers of the Effect of PM_{10} on Hospital Admissions for Heart and Lung Disease

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Studies on acute effects of particulate matter (PM) air pollution show significant variability in exposure–effect relations among cities. Recent studies have shown an influence of ventilation on personal/indoor–outdoor relations and stronger associations of adverse effects with combustion-related particles. We evaluated whether differences in prevalence of air conditioning (AC) and/or the contribution of different sources to total PM_{10} emissions could partly explain the observed variability in exposure–effect relations. We used regression coefficients of the relation between PM_{10} and hospital admissions for chronic obstructive pulmonary disease (COPD), cardiovascular disease (CVD), and pneumonia from a recent study in 14 U.S. cities. We obtained data on the prevalence of AC from the 1993 American Housing Survey and data on PM_{10} emissions by source category, vehicle miles traveled (VMT), and population density from the U.S. EPA. We analyzed data using meta-regression techniques. PM_{10} regression coefficients for CVD and COPD decreased significantly with increasing percentage of homes with central AC when cities were stratified by whether their PM_{10} concentrations peaked in winter or nonwinter months. PM_{10} coefficients for CVD increased significantly with increasing percentage of PM_{10} emission from highway vehicles, highway diesels, oil combustion, metal processing, decreasing percentage of PM_{10} emission from fugitive dust, and increasing population density and VMT/mile^2. In multivariate analysis, only percentage of PM_{10} from highway vehicles/diesels and oil combustion remained significant. For COPD and pneumonia, associations were less significant but the patterns of the assocations were similar to that for CVD. The results suggest that air conditioning and proportion of especially traffic-related particles significantly modify the effect of PM_{10} on hospital admissions, especially for CVD. *Key words*: air conditioning, air pollution, combustion sources, hospital admissions, meta-regression. *Environ Health Perspect* 110:43–49 (2002). [Online 15 December 2001]

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In the last decade, epidemiologic studies have documented associations between particulate matter (PM) air pollution and increases in hospital admissions for respiratory and cardiovascular disease in studies all over the world (*1–4*). The magnitude of the estimated PM_{10} effects, however, has differed substantially among different studies. A recent multicity study, conducted in 14 cities thoughout the United States, has documented significant heterogeneity among the city-specific effect estimates (*5*). This heterogeneity could not be attributed to sociodemographic factors such as the percentage of the population with college education, percentage of unemployment, percentage living below the federal poverty level, or the percentage of the population that was nonwhite (*5*).

Exposure studies have shown an influence of ventilation on personal/indoor–outdoor relations. Sarnat et al. (*6*) found that in a panel of 15 nonsmoking older subjects in Baltimore, Maryland, associations between personal $PM_{2.5}$ and ambient concentrations were strongest for well-ventilated indoor environments and decreased with decreasing ventilation. For sulfate, which can be considered a tracer for particulate matter of ambient origin, correlations between personal and ambient concentrations showed less decrease with decreasing ventilation, producing strong personal–ambient associations even in poorly ventilated indoor environments. Personal to ambient ratios and personal–ambient slopes, however, decreased with decreasing ventilation, both for $PM_{2.5}$ mass and for sulfate, with the personal–ambient regression slopes of poorly ventilated indoor environments being almost half the value of the well-ventilated indoor environment (0.46 vs. 0.83 and 0.39 vs. 0.70 for $PM_{2.5}$ and sulfate, respectively) (*6*).Consistent with these findings, previous studies have also shown that personal and/or indoor concentrations of sulfate are lower and less well correlated with outdoor concentrations for homes with air conditioning (AC) than homes without AC (*7,8*), likely because air conditioned homes typically have lower air exchange rates than homes that use open windows for ventilation (*8,9*). For example, in a study by Suh et al. (*8*), the median air exchange rate, measured over 12-hr daytime periods in 47 homes in State College, Pennsylvania, during the summer of 1991, was about six times higher for non-air conditioned homes compared to air-conditioned homes. In the same study, regression of indoor on outdoor sulfate levels yielded a lower slope in air conditioned homes than in non-air conditioned homes (0.36 vs. 0.78)(*8*). These results suggest that the fraction of $PM_{2.5}$ from ambient origin that penetrates indoors is lower in homes with AC than in homes without AC.

Because people spend most of their time indoors, persons living in homes with AC will, at the same outdoor concentrations, be exposed to lower levels of particles from ambient origin than persons living in homes without AC. Consequently, a change in ambient levels of, for example, 10 μg/m^3 will correspond to a smaller change in personal exposures for subjects living in homes with AC than for subjects living in homes without AC, which should cause concentration–effect relationships to be attenuated. Besides the difference in the levels of exposures, a poorer correlation between personal and ambient levels for subjects living in homes with AC could further attenuate the observed exposure–response relationships through misclassification of exposure.

Several recent studies have suggested that fine particles are more responsible than coarse particles for the observed associations between PM air pollution and health effects (*10–12*). Another recent study suggests that combustion particles from mobile sources and coal combustion sources are specifically associated with increased mortality (*13*). In addition, the traffic-related particles were more strongly associated with cardiovascular deaths (*13*). Recent toxicologic studies also suggest that specific components of concentrated air particles may be responsible for specific biologic responses (*14,15*).

We therefore evaluated whether differences in prevalence of AC and/or the contribution of different combustion sources to total PM_{10} emissions could explain part of the observed variability in exposure–effect relations among different cities.

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Methods

Data collection. We used regression coefficients of the relation between ambient PM_{10} and hospital admissions for chronic obstructive pulmonary disease (COPD), cardiovascular disease (CVD), and pneumonia from a recent study in 14 U.S. cities (Table 1) (*5*). Briefly, daily counts of hospital admissions for CVD [*International Classification of Diseases, 9th Revision* (ICD-9), 390–429], COPD (ICD-9, 490–492, 494–496), and pneumonia (ICD-9, 480–487) (*16*), in persons ≥ 65 years were obtained from 14 cities with extended daily PM_{10} measurements for the period 1985 through 1994. For each city, the associations between hospital admissions and PM_{10} were investigated with a generalized additive robust Poisson regression model. We built a model for each city to allow for city-specific differences. The variables included in each model were season, weather variables (24-hr means of temperature, relative humidity, and barometric pressure) and day of week. Because weather and season vary across the cities, however, the smoothing parameter for each variable was optimized separately in each location. We calculated effects of several lags, including distributed lags (both unconstrained and quadratic). For this study we chose lag 0/1 for CVD and lag 1/2 for COPD and pneumonia because PM effects on these outcomes have generally been most prominent for these lags. Details about the methods used to calculate the city-specific effect estimates are given elsewhere (*5*).

We obtained information about the prevalence of AC from the 1993 American Housing Survey of the United States Census Bureau (*17*), which we used to calculate the percentage of homes with central AC for each metropolitan area. We obtained data on PM_{10} emissions by source category and vehicle miles traveled (VMT) by county from the U.S. EPA emissions and air quality data website (*18*). In addition, we obtained data on population density from the 1990 census (*19*). We calculated the percentages of total PM_{10} emission from total highway vehicles, highway diesels, coal fuel combustion (electric utilities, industrial, and commercial/industrial), oil fuel combustion (electric utilities, industrial, commercial/industrial, and residential), residential wood combustion, metal processing, and fugitive dust by dividing the PM_{10} emissions from these sources by the counties' total PM_{10} emissions. We divided vehicle miles traveled in 1996 and the total population in 1990 by the areas of the respective counties to obtain VMT per square mile and population density.

The seasonal pattern in ambient PM concentrations can differ among different cities, with the highest PM_{10} concentrations occuring in summer in some cities and in winter in other cities. Air exchange rates are generally lower in winter than in summer because people keep their windows closed (*20*), so use of outdoor concentrations could cause higher exposure misclassification in cities with winter peaking PM_{10} concentrations compared to cities with nonwinter peaking PM_{10} concentrations. This could reduce the exposure–response relationship in cities with winter peaking PM_{10} concentrations. Hoek et al. (*21*) suggested an explanation for the lower effect estimates that are generally found in Europe compared to the

Table 1. Mean PM_{10} concentrations in summer and winter and regression coefficients (natural log of relative risk for 1 μg/m³ increase, multiplied by 10,000) of the effect of PM_{10} on hospital admissions for CVD, COPD, and pneumonia in 14 U.S. cities.

		Mean PM_{10} concentration (μg/m³)			CVD, PM_{10} Lag 0/1		COPD, PM_{10} Lag 1/2		Pneumonia, PM_{10} Lag 1/2	
City	County	Summer[a]	Winter[b]	Winter/ summer	Coefficient	SE	Coefficient	SE	Coefficient	SE
Birmingham, AL	Blount, Shelby, Walker, St. Clair, Jefferson	40.0	27.4	0.69	4.2	3.2	–13.2	10.3	6.2	5.8
Boulder, CO	Boulder	26.8	36.3	1.35	16.8	13.9	105.0	37.2	7.6	25.4
Canton, OH	Stark	36.6	25.8	0.70	7.3	6.8	29.7	18.1	11.0	13.7
Chicago, IL	Cook	42.5	30.4	0.71	13.1	1.7	8.8	5.1	13.0	3.0
Colorado Springs, CO	El Paso	21.3	37.3	1.75	11.6	8.9	36.2	22.4	40.8	14.6
Detroit, MI	Wayne	42.8	32.8	0.77	15.1	1.8	22.7	4.9	21.1	3.3
Minneapolis, MN	Hennepin, Ramsey	30.5	23.0	0.75	7.3	3.8	24.8	11.7	27.1	7.0
Nashville, TN	Davidson	40.1	31.9	0.80	2.0	6.0	–5.3	19.1	–14.4	12.7
New Haven, CT	New Haven	30.3	31.6	1.04	21.0	4.2	35.4	17.4	23.6	8.4
Pittsburgh, PA	Allegheny	46.6	29.4	0.63	12.4	1.7	16.6	4.6	11.7	3.5
Seattle, WA	King	23.8	43.3	1.82	10.7	2.6	16.5	8.0	9.8	5.1
Spokane, WA	Spokane	32.7	42.2	1.29	6.0	3.3	6.7	7.7	4.5	4.9
Provo-Orem, UT	Utah	31.4	66.3	2.11	3.5	5.7	–45.9	25.8	0.8	8.8
Youngstown, OH	Columbiana, Mahoning	40.7	30.1	0.74	10.2	6.2	32.6	17.3	6.3	13.5

[a]Average of June, July, and August. [b]Average of January, February, and December.

Table 2. Percentage of homes with central AC, population density, VMT/mile², and percentage of PM_{10} from different sources per city.

		PM_{10} from different sources (%)								
City	Central AC (%)	Highway vehicles	Highway diesels	Coal combustion	Oil combustion	Wood burning	Metal processing	Fugitive dust	Population Density	VMT/ mile²
Birmingham, AL	70.2	1.23	0.74	5.30	0.05	1.06	2.63	81.9	228	3.2
Boulder, CO	6.3	1.36	0.85	0.56	0.06	1.09	0.03	78.6	303	2.5
Canton, OH	29.7	2.34	1.43	0.40	0.13	1.22	1.66	76.4	638	5.8
Chicago, IL	43.2	4.26	2.16	0.44	0.51	0.09	6.82	62.3	5,398	46.4
Colorado Springs, CO	13.4	1.29	0.73	0.48	0.03	0.64	0.07	85.8	187	1.6
Detroit, MI	41.0	6.02	3.06	2.40	0.35	0.57	8.25	54.0	3,439	32.2
Minneapolis–St. Paul, MN	48.4	2.62	1.36	0.44	0.13	0.47	0.30	82.3	2,131	21.1
Nashville, TN	72.2	2.28	1.18	0.27	0.34	1.36	0.02	91.4	1,017	15.6
New Haven, CT	23.9	4.34	2.45	0.00	1.07	26.15	0.06	60.6	1,327	10.4
Pittsburgh, PA	33.4	4.21	2.38	2.64	0.21	0.78	9.05	70.0	1,830	15.5
Seattle, WA	6.2	2.44	1.31	0.05	0.03	3.73	0.36	86.4	709	7.6
Spokane, WA	28.2	1.69	1.00	0.03	0.03	6.13	1.57	59.2	205	1.9
Provo-Orem, UT	26.9	1.58	0.92	0.41	0.24	0.58	4.78	70.4	132	1.4
Youngstown, OH	22.8	2.40	1.53	0.49	0.10	1.95	1.31	83.8	394	3.5

United States: In the United States PM concentrations typically peak in summer whereas in Europe they peak in the winter. To evaluate this, we characterized the cities as either winter or nonwinter peaking. We calculated average PM_{10} concentrations per month using the daily ambient PM_{10} concentrations that were also used to calculate the city-specific effect estimates (*5*). In addition to visual inspection of plots of the specific mean PM_{10} concentrations by month, we calculated the ratios between the mean concentrations during summer (June, July, and August) and winter (January, February, and December).

Statistical analysis. We analyzed the data as a two-stage hierarchic model. The first stage produced the city-specific coefficients published previously (*5*). In the second stage, an ecologic regression was fit where we assumed

$$\beta_i = c_0 + \gamma Z_i + \varepsilon_i,$$

where β_i is the PM_{10} coefficient in city *i*, Z_i are the predictors in city *i*, and we assumed

$$E(\varepsilon_i) = 0 \text{ and}$$

$$Var(\varepsilon_i) = \delta_i^2 + \delta,$$

where δ_i^2 is the within city estimate of the standard error of β_i and δ represents the heterogeneity in the β_i not explained by the predictors *Z*. We estimated c_0 and γ using inverse variance weighted least squares regression. We estimated the between-city variance δ using an iterative maximum likelihood approach (*22*).

Results

Table 2 shows the distributions of the independent variables. The percentage of homes with central AC ranged from 6% in Seattle to 72% in Nashville. On average, 57% of the PM_{10} emissions from highway vehicles came from highway diesels. In total, the different combustion sources on average accounted for about 10% of the total primary PM_{10} emission, whereas on average 74% was from fugitive dust.

Table 3 shows the correlation among the different independent variables. Percentage of homes with central AC was not strongly correlated with any of the combustion-related variables. Percentage of PM_{10} from highway vehicles and highway diesels were highly correlated with population density and VMT per square mile, and also with PM_{10} from oil combustion (*r*, 0.68–0.87). Percentage of homes with AC was not significantly correlated with mean temperature during the study period ($r = 0.13$).

Air conditioning. Five cities (Boulder and Colorado Springs, Colorado; Seattle and Spokane, Washington; and Provo-Orem, Utah) were classified as having winter peaking PM_{10} concentrations, with winter/summer concentration ratios ranging from 1.3 to 2.1 (Table 1). For all cities, central AC was not strongly associated with PM_{10} coefficients (Table 4). However, when we analyzed the data for cities with nonwinter peaking and winter peaking PM_{10} concentrations separately (Table 4, Figure 1), coefficients for CVD-related hospital admissions decreased significantly with increasing percentage of central AC for both nonwinter peaking and winter peaking citites. A model that adjusted for whether the cities were characterized by winter peaking PM_{10} concentrations (yes/no) yielded results that were similar to those of the stratified analysis. For COPD, a significant association was found for the nonwinter peaking cities, whereas for the winter peaking cities a very high but nonsignificant percentage change of 204% was found. As can be seen in Table 1, an important part of the variance between the COPD coefficients was caused by extreme high and low values found for Boulder and Provo-Orem respectively, both of which are winter peaking cities and are also the two smallest cities (total population < 300,000). As a result, the variance in the COPD coefficients for winter peaking cities was much higher than the average within-city variance. Adding the between-city variance component to the weights therefore produced similar weights for all five cities and a very steep slope. When only the within-city variance was included in the weights (fixed effects model), we found an estimated percentage decrease in the COPD coefficients of 89% (SE 96%) per interquartile range increase in central AC, which is similar to the value for nonwinter peaking cities of 82% (Figure 1B).

Coefficients for pneumonia were only marginally significantly associated with percentage of homes with central AC in the model that adjusted for whether the cities were characterized by winter peaking PM_{10} concentrations (yes/no). The pattern of all nonsignificant associations, however, was similar to that found for CVD.

In univariate analyses, we did not find any significant differences between PM_{10} coefficients of nonwinter peaking and winter

Table 3. Spearman correlation matrix.

	PM_{10} from different combustion sources (%)								
Source	Highway vehicles	Highway diesels	Coal	Oil	Wood burning	PM_{10} from metal processing	PM_{10} from fugitive dust (%)	Population density	VMT/mile^2
Central AC	0.15	0.17	0.21	0.42	–0.41	0.28	–0.11	0.43	0.54**
PM_{10} from highway vehicles		0.96#	–0.15	0.68#	–0.11	0.30	–0.50**	0.87#	0.81#
PM_{10} from highway diesels			–0.07	0.69#	–0.02	0.38	–0.57**	0.81#	0.75#
PM_{10} from coal combustion				–0.08	–0.55**	0.42	0.01	0.05	0.04
PM_{10} from oil combustion					–0.29	0.19	–0.38	0.67#	0.66**
PM_{10} from wood burning						–0.47*	0.13	–0.30	–0.31
PM_{10} from metal processing							–0.57**	0.21	–0.18
PM_{10} from fugitive dust								–0.25	–0.16
Population density									0.98#
VMT/mile^2									

*$p < 0.10$. **$p < 0.05$. #$p < 0.01$.

Table 4. Percentage change in the coefficient of the effect of ambient PM_{10} on hospital admissions for CVD, COPD, and pneumonia for an interquartile range increase in the percentage of homes with central AC.

		β CVD, Lag 0/1		β COPD, Lag 1/2		β Pneumonia, Lag 1/2	
Homes with AC	No.	Change (%)	SE (%)	Change (%)	SE (%)	Change (%)	SE (%)
All cities	14	–15.2	14.8	–50.8*	23.5	–16.7	24.4
Nonwinter peaking cities	9	–50.3**	17.4	– 82.1**	25.1	–49.9	32.1
Winter peaking cities	5	–51.7**	13.8	–203.6	118.3	–71.2	74.4
Adjusted for winter peaking PM_{10} (yes/no)	14	–50.5#	0.46	– 91.6#	27.9	–52.8*	28.8

*$p < 0.10$. **$p < 0.05$. #$p < 0.01$.

peaking cities. After adjustment for central AC, however, PM_{10} coefficients of nonwinter peaking cities were significantly higher than those of winter peaking cities for CVD ($p < 0.01$) and COPD ($p < 0.05$) and to a lesser extent for pneumonia ($p = 0.06$) as well.

Mean temperature during the study period was not significantly associated with any of the three hospital admission coefficients.

Source-related variables. Table 5 shows the results of the regression analysis of the source-related variables. All results were adjusted for central AC percentage. The univariate relationship with percentage of PM_{10} from highway vehicles is shown in Figure 2. Coefficients for hospital admissions for CVD increased significantly with increasing percentage of PM_{10} from highway vehicles, highway diesels, oil combustion, metal processing, increasing population density, and VMT per square mile and with decreasing percentage of PM_{10} from fugitive dust. All of these variables were significantly correlated with one another, except metal processing. Given the number of observations available ($n = 14$), the number of independent variables we could simultaneously include in the model was limited. The associations were strongest for PM_{10} from highway vehicles/diesels (highest t-values). When central AC and percentage of PM_{10} from highway vehicles/diesels were included in the model together with one of the other significant variables, all variables except oil combustion lost significance, and the percentage change in PM_{10} coefficient for an interquartile range change in the explanatory variable fell to < 6%. In contrast, the highway and central AC variables remained stable. In the model with oil combustion, the estimated percentages change in PM_{10} coefficient per interquartile range change in the explanatory variable decreased from 56–58% to about 40% (SE 10%) for highway vehicles/diesels and from 38% to 21% (SE 6%) for oil combustion, but both variables as well as the AC variable remained significant.

None of the source-specific variables were significantly associated with coefficients for COPD. As mentioned previously, an important part of the heterogeneity in the COPD coefficients was caused by Boulder and Provo-Orem. When these two cities were excluded from the analysis, the variance in the COPD coefficients decreased by > 75%. Furthermore, the percentage increase in PM_{10} coefficients associated with an interquartile range change in the independent variables became more similar to those for CVD and pneumonia, with a significant association found for percentages of PM_{10} from highway vehicles (percentage change 50%; SE 19%) and highway diesels (percentage change 49%; SE 18%). Coefficients for pneumonia were only marginally significantly associated with percentage of PM_{10} from highway vehicles/diesels, although, as was the case for the associations with AC, the pattern of nonsignificant associations for pneumonia was similar to that for CVD.

Discussion

Air conditioning. Regression coefficients of the relation between ambient PM_{10} and hospital admissions for CVD and COPD of 14 different cities throughout the United States decreased significantly with increasing percentage of homes with central AC. The associations became most apparent when cities were characterized by their PM_{10} concentrations as either nonwinter peaking or winter peaking. We found a similar pattern of associations for pneumonia, although the standard errors of the estimates were larger those for CVD and COPD.

To our knowledge, no other studies have looked at AC use as an effect modifier of the relation between PM_{10} and hospital admissions. In a recent meta-analysis of the time-series PM-mortality literature, Levy et al. (*23*) did include percentage of homes with central AC as one of the variables to explain the variability in effect estimates. With the "t-to-enter" statistic, percentage of homes with central AC did not enter the model, although stratified analyses showed higher effect estimates for cities with < 30% of homes with central AC [effect estimate 0.76% per 10 μg/m^3; confidence interval (CI), 0.54–0.98] than for cities with > 30% of homes with central AC (effect estimate 0.57% per 10 μg/m^3; CI, 0.39–0.74). That

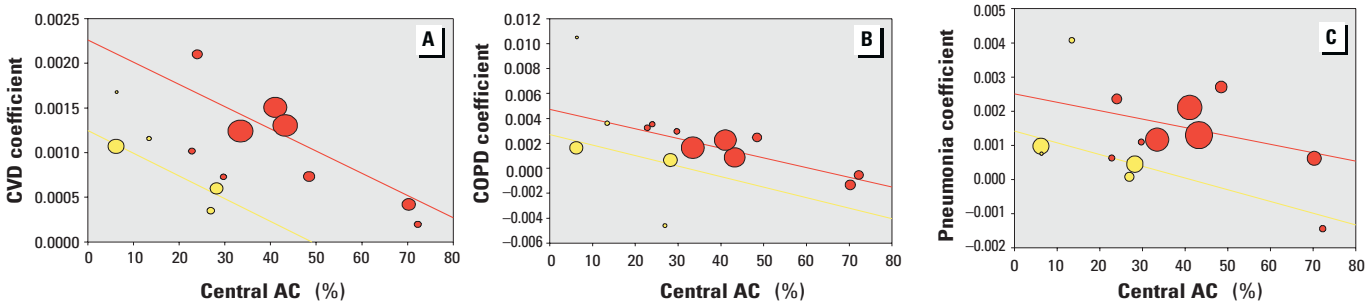

Figure 1. Univariate relation between percentage of homes with central AC and regression coefficients for (*A*) CVD, (*B*) COPD, and (*C*) pneumonia, for cities with nonwinter peaking PM_{10} concentrations (red) and winter peaking PM_{10} concentrations (yellow). Circle area is proportional to the inverse of the variance of the effect estimate. Lines represent inverse variance weighted regression equations (fixed-effects model).

Table 5. Percentage change in the coefficient of the effect of ambient PM_{10} on hospital admissions for CVD, COPD, and pneumonia for an interquartile range increase in the percentage of PM_{10} from different sources, population density, and VMT/mile^2, adjusted for central AC (%).

Source	β CVD, Lag 0/1		β COPD, Lag 1/2		β Pneumonia, Lag 1/2	
	Change (%)	SE (%)	Change (%)	SE (%)	Change (%)	SE (%)
PM_{10} from highway vehicles	58.0**	9.9	48.0	29.7	60.7*	29.5
PM_{10} from highway diesels	55.6**	9.4	47.4	28.2	55.5*	28.7
PM_{10} from coal combustion	0.6	2.6	–0.4	4.7	1.9	5.0
PM_{10} from oil combustion	37.5**	9.3	2.3	24.9	26.5	22.9
PM_{10} from wood burning	2.7	3.2	1.4	5.7	2.3	5.1
PM_{10} from metal processing	29.0**	13.0	2.7	27.3	12.1	29.3
PM_{10} from fugitive dust	–49.4**	16.5	–10.9	36.3	–31.3	37.9
Population density	22.4**	7.8	16.6	19.9	27.6	18.6
VMT /mile^2	21.2**	7.4	16.4	17.9	26.4	17.7

*$p < 0.10$. **$p < 0.05$.

study, however, used 19 effect estimates from 13 different U.S. cities, published in 12 different papers by 6–7 different research groups. As a result, the study incorporated coefficients obtained using a variety of statistical models, including different lag times and control strategies for confounders (*23*). In our study, we used the same statistical methods for all cities and included the same confounding variables in all models. Only the smoothing parameters for each variable were optimized seperately in each location, to allow for city-specific differences (*5*). Moreover, Levy et al. (*23*) did not stratify or control their analysis for winter versus summer peaking PM_{10} concentrations.

Several studies that have included more than one city also found substantial differences in the city-specific effect estimates (*24,25*). For cardiovascular disease, Moolgavkar (*24*) recently documented significant associations between daily hospital admissions and PM_{10} in single-pollutant models in Cook County, Illinois, and Los Angeles County, California, communities with prevalences of central AC of 43% and 31%, respectively. In contrast, the same study found no significant associations in Maricopa County, Arizona, where almost 90% of the homes have central AC. Similarly, for COPD and pneumonia, Moolgavkar et al. (*25*) found significant association between PM_{10} and hospital admissions in Minneapolis–St. Paul, Minnesota, where 48% of the homes have central AC, whereas no effect was found in Birmingham, Alabama, which has a higher prevalence of AC (70%). This between-city variability in the associations is consistent with findings from our study.

The relation between AC and PM_{10} coefficient became more apparent when the cities were classified as having either nonwinter peaking or winter peaking PM_{10} concentrations. The slopes of the relations between PM_{10} coefficients and percentage of homes with central AC were similar for nonwinter and winter peaking cities, but the y-intercepts for the nonwinter peaking cities were higher than those for the winter peaking cities. The difference in the intercepts implies that, for cities with no AC, the effect of PM_{10} is stronger in cities with nonwinter peaking concentrations than for cities with winter peaking concentrations. This may be explained by the fact that air exchange rates are generally lower in winter than in summer because people keep their windows closed (*20*). The fraction of ambient particles that penetrates indoors is therefore probably lower in winter than in summer, producing a smaller increase in indoor and personal concentrations per increase in ambient concentrations in winter, especially for homes without AC. Although the similarity of the slopes suggests that the stronger effect in nonwinter peaking than in winter peaking cities is also present between any two cities with the same percentage of homes with AC, the relatively low percentages of AC in the winter peaking cities (< 30%) make it difficult to predict to what extent the difference found in our study can be extrapolated to winter peaking cities with higher percentages of homes with AC. Both the decrease in PM_{10} coefficients in locations with more central AC and the lower PM_{10} coefficients in locations with winter peaking particle concentrations support the conclusion that factors that reduce the slope between outdoor PM and indoor PM of ambient origin also reduce the slope of the association between ambient PM_{10} and health outcomes. This also supports the causality of the exposure–effect associations, because if outdoor PM_{10} were a surrogate for something else, such as seasonality or epidemics, this pattern across cities would not be expected.

The associations observed with AC could also be related to other city-specific characteristics associated with percentage of homes with AC. Because of the limited sample size (14 cities), the extent to which we could adjust for potential confounders was limited. Because use of AC is related to temperature, temperature would be the most obvious potential confounder. The model we used to calculate the city-specific PM_{10} regression coefficients in the first stage of the analysis, however, included variables for 24-hr means of temperature, relative humidity, and barometric pressure, among others, and the smoothing parameters for these variables were optimized separately in each location. The resulting city-specific regression coefficients are therefore already adjusted for the effect of day-to-day variations in temperature (and other weather variables), so the results of our meta-regressions (second stage) are not likely to be confounded by differences in the temporal patterns of these meteorologic conditions. Because mean temperature during the study period was not significantly correlated with percentage of homes with AC ($r = 0.13$) and no significant associations between mean temperature and any of the hospital admission coefficients were found, our results are also not likely to be confounded by long-term differences in temperature between the cities.

Source-related variables. PM_{10} regression coefficients for CVD increased significantly with increasing percentage of PM_{10} emission from highway vehicles, highway diesels, oil combustion, and metal processing, decreasing percentage of PM_{10} emission from fugitive dust, and increasing population density and VMT per square mile. In multivariate analysis, however, only percentages of PM_{10} from highway vehicles/diesel and oil combustion were significantly related to coefficients for CVD. None of the variables were significantly associated with hospital admissions for COPD, and hospital admissions for pneumonia were only marginally significantly associated with percentage of PM_{10} emission from highway vehicles/diesels. As was the case for percentage of homes with central AC, however, the patterns and magnitude of the nonsignificant associations for COPD and pneumonia were similar to those for CVD. Furthermore, when Boulder and Provo-Orem were excluded from the analysis for COPD, associations for motor vehicle-related PM_{10} became significant.

Source-specific morbidity effects are consistent with findings from particle mortality and toxicology studies. Using factor analysis

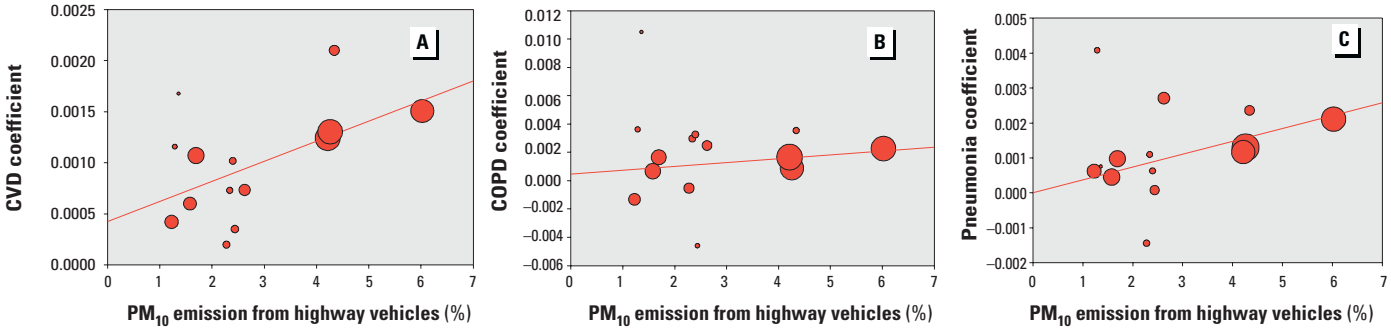

Figure 2. Univariate relation between percentage of PM_{10} from highway vehicles and regression coefficients for (*A*) CVD, (*B*) COPD, and (*C*) pneumonia. Circle area is proportional to the inverse of the variance of the effect estimate. Lines represent inverse variance weighted regression equations (fixed-effects model).

techniques, Laden et al. (*13*) also found significant associations between several distinct combustion source-related fractions, determined using the elemental composition of $PM_{2.5}$ samples, and daily mortality in six eastern U.S. cities. A 10 μg/m^3 increase in $PM_{2.5}$ from mobile sources accounted for a 3.4% increase in daily mortality, with the strongest associations found for deaths caused by ischemic heart disease compared to those caused by COPD or pneumonia. In comparison, the same 10 ug/m^3 increase from coal combustion sources produced a smaller mortality increase of 1.1%, with the strongest associations found for respiratory deaths. For PM_{10} from oil combustion, associations were less significant, but even larger per microgram per cubic meter, with a nonsignificant summary effect of 5.6%. No associations were found for $PM_{2.5}$ from crustal particles (*13*). These results are consistent with our findings of greater-than-average impacts of traffic particles, particularly for heart disease, greater impact of oil combustion particles, and smaller effects of fugitive dust. Source-related fine particles were also significantly associated with subtle alterations in pulmonary and systemic cell profiles in canines exposed to concentrated air particles (*14*). Among other source-related effects, combustion-related metals were linked to peripheral blood parameters, and coal-related particles were related to hematologic alterations.

It is interesting to note that in both our study and Laden et al.'s (*13*) mortality study, the source-specific associations were strongest for vehicle exhaust-related particles. In our study, highway diesels comprised over 50% of the total percentage of PM_{10} emission from highway vehicles. Because these two variables were very highly correlated (Spearman $r = 0.96$), our data do not allow any conclusions about the relative importance of diesel exhaust particles compared to other vehicle exhaust particles. Several studies, however, have suggested that diesel exhaust particles in particular are associated with health outcomes (*26,27*). An epidemiologic study in the Netherlands showed that exposure to diesel exhaust particles, measured as either truck traffic density or classroom black smoke concentrations, was significantly associated with reduced lung function and increased prevalence of chronic respiratory symptoms in school children (*26,27*). In addition, deaths from cardiovascular disease in European cities have been associated with ambient concentrations of black smoke, for which the major source is diesel-powered vehicles, with the effect estimates of black smoke being more stable than those for PM_{10} (*21,28*).

Of the source-specific parameters, only PM_{10} emissions from fugitive dust were negatively associated with coefficients for CVD. Because PM_{10} from fugitive dust consists primarily of coarse mode particles, this finding is consistent with the observed importance of motor vehicle-related and oil combustion particles, which are primarily fine particles, and with the growing evidence of stronger associations for fine than for coarse particles (*10–12,23*).

For each of the source-specific parameters, associations were strongest for CVD, suggesting that the observed relations may be disease specific. Because the patterns of the associations were generally similar for all three disease categories, however, results may also be related to the power of the statistical analyses, where hospital admission rates for CVD are much higher than those for COPD and pneumonia. For the cities and time period included in this study, mean daily Medicare admissions for CVD were over 10 times higher than those for COPD and almost four times higher than those for pneumonia (*5*). As a result, the PM_{10} effect estimates for COPD and pneumonia are more subject to random error than those for CVD, which could explain the higher standard errors of our meta-regression results for COPD and pneumonia.

One of the limitations of this study is that we relied on U.S. EPA emissions summaries to characterize the contribution of different sources to total PM_{10} emissions. These estimates may not be the best indicators of the actual differences in the composition of the PM mixtures. For the traffic-related particles we also evaluated two other measures of exposure—vehicle miles traveled per square mile and population density—which were highly correlated with each other and also with the highway emission variables (Spearman r, 0.74–0.98). These alternative variables also yielded significant associations with coefficients for cardiovascular disease. Although these associations lost significance when entered in the model together with the U.S. EPA estimate, they do provide additional support for a stronger effect of traffic-related particles on hospital admissions for cardiovascular events in particular. The greater explanatory power of the U.S EPA estimates of percentage of PM_{10} emission from traffic or diesel than that of the VMT and population density variables provide some assurance that the EPA estimates are reasonable.

Conclusions

This study suggests that while sociodemographic factors previously have explained little of the variation in city-specific coefficients for PM_{10}, exposure-related differences could potentially explain a substantial fraction of that variability. In particular, the variability in the observed effects of ambient PM_{10} on hospital admissions for heart and lung disease among different cities was attributed to differences in percentage of homes with central AC and percentage of PM_{10} emissions from specific combustion sources, especially for CVD. Because of the ecologic nature and the limited sample size of our study, the role of central AC as a modifier of the effect of PM_{10} and the potentially higher toxicity of particularly traffic-related particles must be further investigated.

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