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AIRBORNE COARSE PARTICLES AND MORTALITY

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Some recent epidemiologic studies suggest a stronger effect of fine particles (PM2.5) than of coarser particulate matter. To examine the support for such a differential effect, the authors conducted a daily time-series analysis of mortality in relation to measurements of PM2.5, PM10, and PM10-2.5 in southwestern Mexico City in the years 1992–1995. A generalized linear model based on Poisson regression was used to control for weather and periodic cycles, and the average concentration of the previous five days was the index of particle exposure. The mean concentrations of PM2.5 and PM10 were 27.4 μg m^{-3} and 44.6 μg m^{-3}, respectively, and the mean concentration of PM10-2.5 was 17.2 μg m^{-3}. PM10 was highly correlated with both the fine and coarse fractions, but PM2.5 and PM10-2.5 were rather weakly correlated with each other (correlation coefficient 0.52). All three particle size fractions were associated individually with mortality: a 10-μg m^{-3} increase in PM10

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was associated with a 1.83% increase in total mortality (95% CI −0.01–2.96), and an equal increment in PM2.5 was associated with a 1.48% increase in deaths (95% CI 0.98–2.68%). The largest effect was observed for a 10-μg m^{-3} increment in PM10-2.5; mean daily mortality increased 4.07% for each 10 μg m^{-3} (95% CI 2.49–5.66%). The effect of coarse particles was stronger for respiratory diseases than for total mortality, cardiovascular diseases, or other noninjury causes of death. These patterns persisted after adjustment for O_3 and NO_2. When both PM2.5 and PM10-2.5 were included simultaneously in the regression model, the effect of PM10-2.5 remained about 4% per 10 μg m^{-3} (95% CI 1.96–6.02%), while the effect of PM2.5 was virtually eliminated (0.18% change). These associations may be attributable to specific combustion or biogenic materials within the coarse particle mass. Understanding these relationships will require analyses of the composition of coarse particles. The findings also suggest a need to that the relative effects of coarse and fine particles on mortality should be examined in more cities with a wider variety of climates, population characteristics, and air pollutants.

Epidemiologic studies around the world show that short-term increases in the level of ambient airborne particles are associated with acute increases in mortality (Schwartz, 1994; U.S. EPA, 1996; Thurston, 1996; Zmirou et al., 1998). While the consistency of this association is remarkable, the mechanism that may be responsible for it is not clear. Because airborne particulate matter is not uniform, an important question is whether health effects vary by particle size, origin, composition, or morphology.

Recent research (Dockery et al., 1993; Pope et al., 1995; Schwartz et al., 1996) has focused attention on the role of fine particles (PM2.5) in producing the observed effects associated with airborne particles. Our group previously reported effects of fine particles on daily mortality in adults and infants in Mexico City (Borja-Aburto et al., 1998; Loomis et al., 1999). To examine the support for a difference in response according to particle size, we reanalyzed a time series of air pollution and mortality data in Mexico City from 1993 to 1995.

METHODS

The research area and methods have been described in a previous publication (Borja-Aburto et al., 1998). Briefly, the study was conducted in a 240-km^2 urban area with about 2.5 million residents formed by 6 political jurisdictions in the southwest part of the Federal District of Mexico. We obtained electronic records from death certificates of residents of this area in the years 1993–1995, and excluded deaths from external causes (accidents, poisoning and violence) and deaths that occurred outside the Federal District. The remaining causes of death were coded according to the 9th revision of the International Classification of Diseases (ICD). We reduced the death data to daily counts for all ages, all nonexternal causes, plus deaths from respiratory (ICD-9 codes 460–466, 480–508), and cardiovascular disease (ICD-9 codes 390–417, 420, 430–448).

Ambient air pollutant levels were monitored at a station operated by Universidad Autonóma Metropolitana-Xochimilco and the Harvard School of Public Health, with support from the U.S. Environmental Protection Agency (EPA). The monitoring station was located at a primary school in the study area. Levels of inhalable particles (PM10) and fine particles (PM2.5) were recorded as 24-h integrated mass concentration, with samples collected on Teflon filters using Harvard impactor low flow size-fractionated particle samplers. Filters were collected daily at 8 a.m. and particle mass was determined gravimetrically in a temperature- and humidity-controlled laboratory, according to a standard, written protocol. Coarse-particle mass was estimated as the difference between PM10 and PM2.5. Concentrations of O_3, NO_2, and SO_2 were measured hourly, using U.S. EPA reference methods: ultraviolet photometry for ozone, chemiluminescence for nitrogen oxides, and pulsed fluorescence for SO_2.

Instruments were calibrated bimonthly, and external audits of all monitoring systems were performed twice a year by the Harvard School of Public Health, Boston. Pollutant measurements that failed to meet quality assurance criteria were excluded from the epidemiologic analysis.

José Camacho Salazar (personal communication) provided meteorological data from a station operated by the Observatorio Meteorológico del Colegio de Geografía of the Universidad Nacional Autónoma de México.

We used Poisson regression to model daily mortality, employing a generalized additive model, to allow for both linear and nonlinear relationships between mortality and predictor variables (Hastie & Tibshirani, 1990). Because the function governing the behavior of mortality over time is unknown, we developed nonparametrically smoothed, empirical functions of predictor variables empirically using LOESS, a standard smoothing algorithm. To account for the potential effects of serial correlations among observations, we multiplied the estimated variance of the regression coefficients by the square root of the overdispersion parameter (McCullagh & Nelder, 1989) to adjust for extra-Poisson variation.

Before considering air pollutants, we developed a basic model for mortality. We examined smoothed functions of time and weather indicators with varying time lags and averaging periods. In comparing regression models, we evaluated both the magnitude of the coefficients and goodness of fit, assessed by Akaike's Information Criterion (AIC), a measure of model deviance adjusted for the number of parameters (Hastie & Tibshirani, 1990). Details of model development have been reported elsewhere (Borja-Aburto et al., 1998; Loomis et al., 1999). The mortality model that provided the best compromise between fit and number of parameters contained a smoothed term for time with 7.7 degrees of freedom and the mean temperature during the 3 days before death (Loomis et al., 1999).

We added pollution variables to the basic mortality model. Preliminary graphical analyses indicated that the relationship of particles and mortality

was approximately linear, so particle concentrations were entered as continuous variables scaled in micrograms per cubic meter. A similar approach was used for gases, with the pollutant variables scaled in parts per billion. Particles were of primary interest, and gaseous pollutants were evaluated as potential confounders of the association of particles with mortality; details concerning the associations of ozone and NO_2 with mortality have been published previously (Borja-Aburto et al., 1998). Sulfur dioxide was not considered because the concentrations were negligible.

The association of particles with mortality was expressed as percent change per 10 $\mu g\ m^{-3}$ by multiplying by 1000 the coefficient of particles in the Poisson model. We present the effect on mortality of an absolute increment in pollutant concentration rather than per interquartile range (IQR), as some other authors have done, because in our data the IQRs were equal for PM2.5 (IQR 14) and PM10-2.5 (IQR 13), and twice as large for PM10 (IQR 24).

To account for the possible time lag between exposure and the appearance of effects and to examine the effect of mean exposures during extended periods, we considered exposures to each pollutant within windows of 1 to 4 days of duration, beginning from 0 to 5 days before death.

RESULTS

On average, there were 32 deaths per day, of which 9 were from cardiovascular causes and 3 were from respiratory causes (Table 1). The average concentration of fine particles (PM2.5) was 27.4 $\mu g\ m^{-3}$ and the mean concentration of total inhalable particles (PM10) was 44.6 $\mu g\ m^{-3}$, while the mean concentration of the coarse fraction of inhalable particles (PM10-2.5) was 17.2 $\mu g\ m^{-3}$ (Table 1). With the exception of ozone, the levels of gaseous pollutants were generally low (Table 1).

The level of particulate matter had strong seasonality, with higher concentrations from December to April, and lower concentrations during the rainy season from May to November (Figure 1). On average, 62% of PM10 was composed of particles less than 2.5 μm in diameter. However, the ratio of PM2.5/PM10 varied from 80% during the rainy season to 50% during the dry season (Figure 1). The coefficients of variation of the three components of particulate matter were 0.38 for PM2.5, 0.38 for PM10, and 0.50 and for PM10-2.5. PM10 was highly correlated with both the fine and coarse fractions (correlation coefficients .89 and .84, respectively), but PM2.5 and PM10-2.5 were only moderately correlated (coefficient .52).

The lag structure of the relation of particle exposure and mortality was evaluated using 1-day exposure windows (Figure 2). When longer exposure windows were examined, the average exposure during the previous 5 days showed a stronger and more precise association (Table 2). We used the average concentration of the previous 5 days as the index of particle exposure in subsequent analyses.

TABLE 1. Summary Statistics for Mortality, Air Pollutant Concentrations, and Weather Indicators, Southwest Mexico City, 1993–1995.

	Total mortality	Cardiovascular diseases	Respiratory diseases	Age ≥65 yr	PM2.5 ($\mu g\ m^{-3}$)	PM10 ($\mu g\ m^{-3}$)	PM10-2.5 ($\mu g\ m^{-3}$)	O_3 (ppb)	NO_2 (ppb)	Minimum temperature (°C)
Valid observations	942	942	942	942	866	866	866	901	861	942
Mean	32	9.1	3.2	17.8	27.4	44.6	17.2	44.0	37.7	9.1
Standard deviation	6.4	3.2	1.9	4.7	10.5	16.8	8.7	15.7	11.4	3.3
Minimum	16	1	0	4	4	10	1	4.1	12.8	−1.2
Lower quartile	27	7	2	15	20	32	10	33.7	29.1	6.7
Median	32	9	3	17	26	43	16	43.7	36.3	9.5
Upper quartile	51	11	4	21	34	56	23	54.2	44.0	12.0
Maximum	55	20	11	36	85	121	55	127.1	86.8	17.2

Note. Deaths, pollutant levels, and meteorological parameters expressed as 24-h means except as noted; observed pollutant levels given under local conditions.

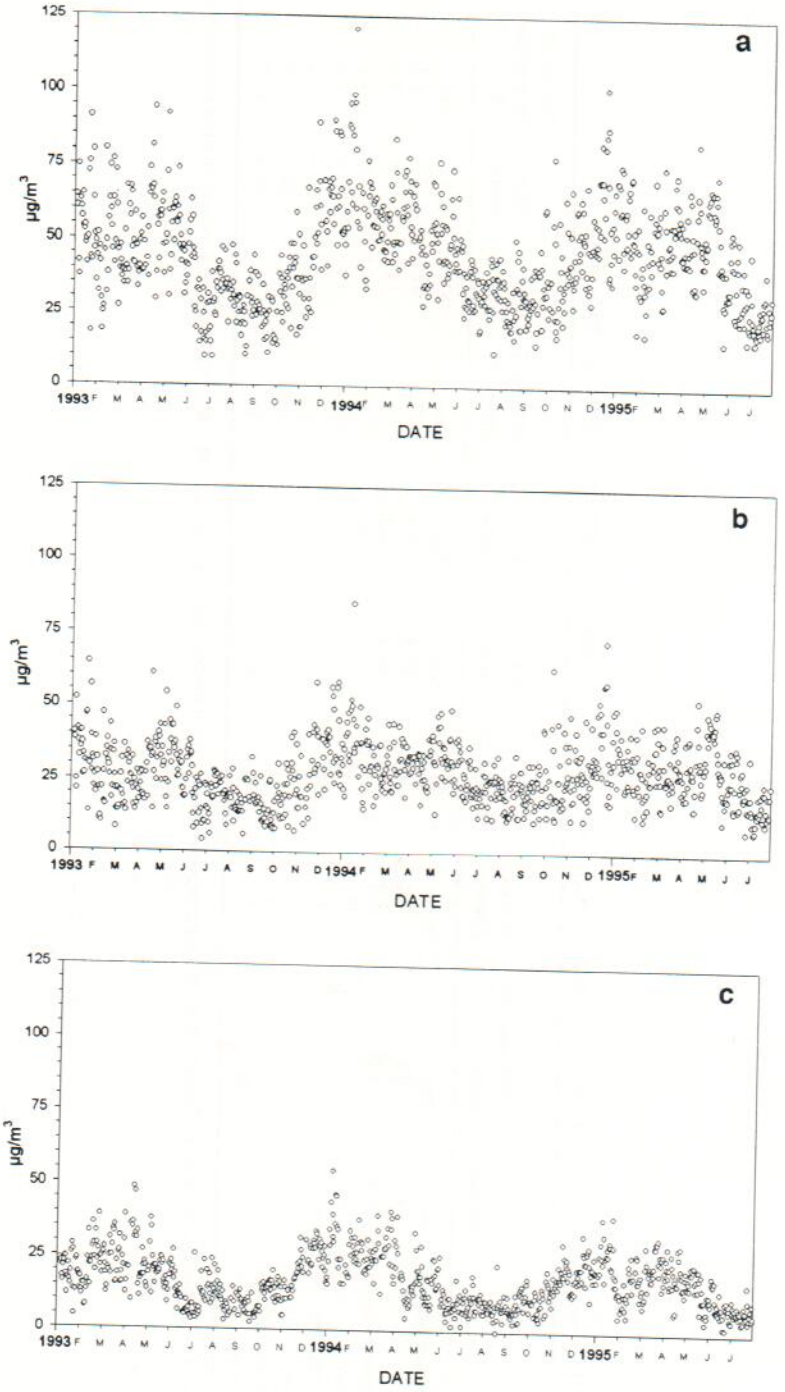

FIGURE 1. (a) Daily mean concentration of PM10 (µg m^{-3}), Mexico City study area, 1993–1995. (b) Daily mean concentration of PM2.5 (µg m^{-3}), Mexico City study area, 1993–1995. (c) Daily mean concentration of PM10-2.5 (µg m^{-3}), Mexico City study area, 1993–1995.

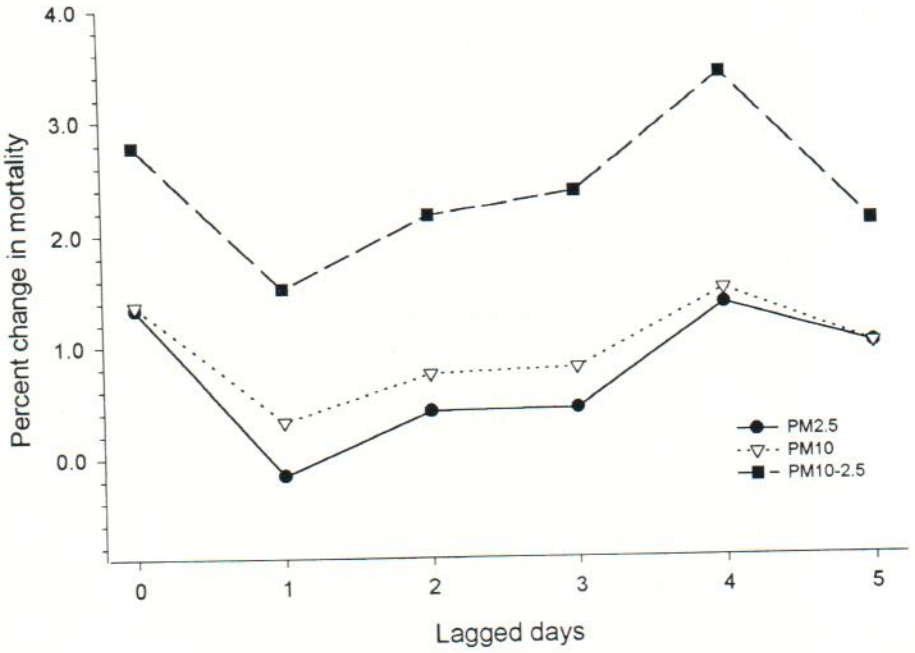

FIGURE 2. Percent increase in total mortality per 10-μg m^{-3} increase in 24-h mean PM2.5, PM10, and PM10-2.5 with lags of 0–5 days.

All three particle-size fractions were associated with mortality (Table 2). Each 10-μg m^{-3} increase in 5-day mean of PM10 was associated with a 1.83% increase in total mortality, and an equal increment in PM2.5 was associated with a 1.48% increase in deaths. The largest effect was observed for a 10-μg m^{-3} increment in PM10-2.5: 4.07% in all-cause mortality (Table 2). Season-specific analyses yielded similar associations for coarse particles in both the dry and rainy season, but the precision of all regression coefficients was reduced (data not shown).

The effect of each type of particle was larger for respiratory diseases than for total mortality, cardiovascular disease mortality, or mortality among persons aged 65 yr or over, and each effect was stronger for coarse particles than for fine particles or PM10 (Table 2). The magnitude of these associations did not change substantially after adjusting for ozone and nitrogen dioxide. Adjustment for ozone did, however, reduce associations of particles with cardiovascular disease mortality (Table 2).

To assess the independent contribution of particles of different sizes to daily mortality, we considered PM2.5 and PM10-2.5 simultaneously in the regression model. The effect of PM10-2.5 remained the same, while the effect of PM2.5 became markedly weaker (Table 3).

TABLE 2. Percent Increase of Cause- and Age-Specific Mortality Associated with a 10-μg m^{-3} Increment in PM2.5, PM10, and PM10-2.5, in Different Models (Estimated by Poisson Regression Controlling for Temperature on the 3 Days Before Death and Nonparametrically Smoothed Periodic Cycles)

	Particle size fraction[a]								
	PM2.5			PM10			PM10-2.5		
	%	95%CI	*t*	%	95%CI	*t*	%	95%CI	*t*
One-pollutant models									
Total mortality	1.48	−0.01, 2.96	1.95	1.83	0.98, 2.68	4.21	4.07	2.49, 5.66	5.03
Respiratory causes	3.6	−1.06, 8.27	1.51	3.85	1.16, 6.55	2.81	8.03	3.05, 13.01	3.16
Cardiovascular causes	1.55	−1.25, 4.35	1.09	2.00	0.39, 3.60	2.44	4.54	1.55, 7.52	2.98
Age >65 yr	1.35	−0.64, 3.35	1.33	2.01	0.86, 3.16	3.43	4.84	2.71, 6.97	4.45
Ozone adjusted[b]									
Total mortality	1.28	−0.43, 2.99	1.47	1.85	0.82, 2.89	3.51	4.28	2.40, 6.15	4.47
Respiratory causes	4.97	−0.35, 10.29	1.83	5.26	2.01, 8.51	3.17	9.99	4.12, 15.86	3.33
Cardiovascular causes	0.44	−0.46, 1.34	0.96	1.29	−0.66, 3.24	1.29	3.58	0.04, 7.11	1.98
Age >65 yr	0.96	−1.35, 3.26	0.81	1.90	0.50, 3.30	2.67	4.78	2.25, 7.31	3.71
Ozone and NO_2 adjusted[b]									
Total mortality	1.25	−1.13, 3.63	1.03	2.47	1.14, 3.81	3.63	4.48	2.44, 6.51	4.32
Respiratory causes	4.57	−2.88, 12.02	1.20	6.40	2.16, 10.64	2.96	9.79	3.42, 16.17	3.01
Cardiovascular causes	0.59	−3.89, 5.06	0.26	1.96	−0.56, 4.48	1.53	3.91	0.08, 7.74	2.00
Age >65 yr	1.60	−1.61, 4.81	0.98	3.07	1.27, 4.87	3.35	5.37	2.64, 8.11	3.85

Note. *t* is the value of the *t* test statistic for the statistical significance of the effect.
[a]Exposure index, mean concentration during the previous 5 days.
[b]Exposure index, 24-h mean for ozone and NO_2.

TABLE 3. Percent Increase in Daily Mortality for Exposure to PM2.5 and PM10-2.5 When Evaluated Simultaneously in a Multivariate Model (Estimated by Poisson Regression Controlling for Temperature on the 3 Days Before Death and Nonparametrically Smoothed Periodic Cycles)

	PM2.5[a]			PM10-2.5[a]		
Outcome	%	95%CI	*t*	%	95%CI	*t*
Total mortality	0.18	−1.72, 2.08	0.18	3.99	1.96, 6.02	3.86
Respiratory	1.15	−4.72, 7.03	0.30	7.46	1.20, 13.73	2.34
Cardiovascular	0.18	−3.40, 3.76	0.10	4.45	0.60, 8.27	2.29
Age >65 yr	−0.37	−2.91, 218	−0.28	5.06	2.35, 7.78	3.66
Others	−0.01	−2.43, 2.41	−0.01	2.98	0.39, 5.57	2.25

[a]Exposure index, mean concentration during the previous 5 days.

DISCUSSION

Our analyses indicate a stronger association of mortality with coarse inhalable particles than with fine particles. While the effects we observed from PM10 and PM2.5 were similar in magnitude to those seen in other cities (U.S. EPA, 1996; Thurston, 1996; Schwartz, 1994; Dockery & Pope, 1994), the effect was stronger for PM10-2.5 than for either of these standard indicators of particle concentration.

These results differ from the principal findings reported by Schwartz and colleagues (1996) in a study of six U.S. cities: In a pooled analysis of these communities, mortality increased 1.5% for each 10 μg m^{-3} of fine particles, and 0.8% for each 10 μg m^{-3} of total thoracic particles (PM10). Nevertheless, while previous studies have suggested that daily mortality is not strongly associated with coarse particle concentrations in general, there is evidence of associations with specific types of coarse particles.

Although Schwartz and colleagues (1996) reported no consistent association with coarse particles in the pooled analysis across all communities, positive associations were found in Steubenville (2.4% increase per 10 $\mu g/m^3$ CM, 95% CI 0.5% to 4.3%, $p = .15$) and in Knoxville (1.0% per 10 $\mu g/m^3$ CM, 95% CI −0.6% to 2.6%, $p = .23$). Both of these locations are dominated by large local sources of coarse combustion particles: steel mills and coal-fired power plants in Steubenville, and coal-fired power plants in the Knoxville area. Thus, coarse combustion particles were associated with increased daily mortality in these two cities.

On the other hand, a negative association with coarse particles was found in Topeka (−1.3% per 10 $\mu g/m^3$ CM, 95% CI −3.3% to 0.6%, $p = .19$), an area in which the coarse particle loadings are dominated by wind-blown crustal materials (Spengler & Thurston, 1983). Schwartz and colleagues (1999) recently found daily mortality was not associated with coarse particles during dust storms in Spokane, WA. Thus, the evidence to date suggests that wind-blown crustal particles are relatively innocuous.

This is not to suggest that all dust in innocuous, however. Biogenic materials in wind-blown dust can be a particular hazard. For example, coccidiodomycosis, commonly known as Valley Fever, is caused by inhalation of spores resuspended from the soil in semiarid areas of the southwestern United States, Mexico, and parts of Central and South America. While the incubation period for coccidioidomycosis is longer than the observed lags for coarse particle associations reported here, it illustrates the potential for biogenic materials in the coarse particle fraction to produce serious health effects.

The health effects of particles of different sizes may differ between cities as a result of variation in particle origin, chemical composition, and biogenic content. Although the characteristics of particles in Mexico City have not been fully examined, several reports suggest that PM10 from the area contains substantial amounts of biogenic material, including aeroallergens and enteric bacteria (Rosas et al., 1994, 1998). A recent study on the effect of Mexico City PM10 on the induction of the lung myofibroblast platelet-derived growth factor receptor system suggests toxic effects of these particles related to both metals and endotoxins (Bonner et al., 1998). The biological components are mainly present in the coarse mass fraction of the particles. Some other laboratory studies suggest that metals may play a role in the generation of physiologic changes, inflammatory responses, or tissue injuries to the cardiorespiratory system, but the metals examined to date have been associated primarily with fine particles (Dreher et al., 1997; Costa & Dreher, 1997).

Limitations of this study include the relatively short span of the time series, the absence of individual measurements of air pollution and other exposures, and the limited information available from death certificates. The statistical power of the study was generally adequate despite the time span because of Mexico City's large population, but reduced numbers precluded some subgroup analyses. In addition, we could not assess the long-term health consequences of air pollution, and, as with other longitudinal studies, the extent to which the study period is typical of events in general cannot be assessed directly.

Lack of individual exposure information is a concern in all studies that employ aggregate data to investigate individual-level causal hypotheses. However, the longitudinal design of this study allows individuals to serve, in effect, as their own controls, thus compensating for the lack of individual information about causes of mortality that are stable from day to day, such as smoking, social class, and occupation.

For air pollution, the correspondence between aggregate measurements from an outdoor stationary monitor and personal exposure depends on the geographic variability of air pollution levels and differences between outdoor and indoor pollutant levels. Wilson and Suh (1997) hypothesized that stationary monitoring data might approximate personal exposure more closely for fine particles than for coarse particles because coarse particles tend to be more strongly influenced by local sources and are less likely to penetrate indoors. They cautioned, however, that their models were based largely on observations in the eastern United States and might not apply in areas with

drier climates (Wilson & Suh, 1997). Some characteristics of our study area may alleviate concerns about the use of outdoor monitoring data. In Mexico City, the differential between outdoor and indoor pollutant levels may be reduced because space heating and air conditioning are seldom used and windows are usually kept open. The geographic variability of various particle size fractions has not been systematically assessed in Mexico City, but the study area in the southwest of the city is distant from major point sources of both industrial and crustal dust, which are concentrated in the northeastern zone of the city. Our measurements confirm that the fine fraction dominates PM10 in the southwest, in contrast to the northeast where the PM2.5:PM10 ratio was 0.47 (unpublished data). Other data suggest greater equilibrium between indoor and outdoor PM10 concentrations in the southwest than in the northeast (indoor/outdoor ratios 1.16 and 1.38, respectively) (CENICA, personal communication).

The design of the current study also helps to control the effect of errors in measuring air pollution exposure: Because all deaths occurring on a given day are assigned a common exposure value, the measurement error is essentially of the Berkson type, which does not bias exposure–disease associations if it is nondifferential with respect to disease occurrence (Armstrong, 1990).

The quality of death certification data in Mexico City has been examined previously and has been found to be good overall; virtually all deaths are physician certified, and the majority occur in a hospital or while under medical care (Loomis et al., 1996). The death certificate database we used for this study includes all deaths of residents of the study area, but we restricted the analysis to deaths occurring in the Distrito Federal to exclude people who may have been out of the city when the observed pollution levels occurred. However, we were not able to consider indicators of potential susceptibility to air pollution, other than age, or to examine deaths by place of occurrence.

Despite their different patterns of deposition, both PM2.5 and PM10-2.5 consist of thoracic particles and have the potential to produce health effects. The associations we observed may be attributable to specific combustion or biogenic materials within the coarse particle mass. Understanding of these relationships will require analyses of the composition of coarse particles. Other factors that should be considered in comparing our findings to other studies include differences in population age structure, underlying health status, and access to medical attention. Mexico City's population is young and consequently has low crude mortality, but a substantial proportion live under impoverished conditions. Our findings therefore suggest that the relationships of coarse and fine particles to mortality should be examined in more cities with a wider variety of climates, population characteristics, and air pollutants.

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