

Associations between Air Pollution and Mortality in Phoenix, 1995–1997

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We evaluated the association between mortality outcomes in elderly individuals and particulate matter (PM) of varying aerodynamic diameters (in micrometers) [PM_{10} , $PM_{2.5}$, and PM_{CF} (PM_{10} minus $PM_{2.5}$)], and selected particulate and gaseous phase pollutants in Phoenix, Arizona, using 3 years of daily data (1995–1997). Although source apportionment and epidemiologic methods have been previously combined to investigate the effects of air pollution on mortality, this is the first study to use detailed PM composition data in a time-series analysis of mortality. Phoenix is in the arid Southwest and has approximately 1 million residents (9.7% of the residents are > 65 years of age). PM data were obtained from the U.S. Environmental Protection Agency (EPA) National Exposure Research Laboratory Platform in central Phoenix. We obtained gaseous pollutant data, specifically carbon monoxide, nitrogen dioxide, ozone, and sulfur dioxide data, from the EPA Aerometric Information Retrieval System Database. We used Poisson regression analysis to evaluate the associations between air pollution and nonaccidental mortality and cardiovascular mortality. Total mortality was significantly associated with CO and NO_2 ($p < 0.05$) and weakly associated with SO_2 , PM_{10} , and PM_{CF} ($p < 0.10$). Cardiovascular mortality was significantly associated with CO, NO_2 , SO_2 , $PM_{2.5}$, PM_{10} , PM_{CF} ($p < 0.05$), and elemental carbon. Factor analysis revealed that both combustion-related pollutants and secondary aerosols (sulfates) were associated with cardiovascular mortality. **Key words:** cardiovascular, composition, factor analysis, particulate matter, $PM_{2.5}$, PM_{10} , sources. *Environ Health Perspect* 108:347–353 (2000). [Online 25 February 2000] <http://ehpnet1.niehs.nih.gov/docs/2000/108p347-353mar/abstract.html>

The associations between air pollution, especially particulate matter (PM), and adverse human health effects have been well documented (1–10). PM is associated with decreased respiratory function, aggravation of existing respiratory and cardiovascular conditions, altered defense mechanisms, and even premature death. The most susceptible populations include those with preexisting respiratory or cardiovascular conditions, asthmatics, children, and the elderly (8,11).

To date, few epidemiology studies have used PM measures other than size-segregated mass as the exposure metric. Schwartz et al. (12) looked at episodes of high coarse particle concentration in Spokane, Washington, and found that windblown dust episodes were not associated with increased mortality. In the Harvard Six Cities Study, Schwartz et al. (3) found a significant association between nonaccidental mortality and particulate matter ≤ 2.5 μm in aerodynamic diameter ($PM_{2.5}$) and sulfur. They did not find a significant association with particulate matter ≤ 10 μm in aerodynamic diameter (PM_{10}) or the coarse fraction of PM [PM_{CF} (PM_{10} minus $PM_{2.5}$)]. In contrast, Ostro et al. (13) found that PM_{10} dominated by coarse particles was associated with an increase in mortality in the Coachella Valley in California. The differences in the results from these two studies may be due to the particulate composition as well as the difference in the amount of PM_{CF} . In the eastern United States, $PM_{2.5}$

is dominated by sulfates (34%), whereas in the western and central United States it is dominated by organic carbon (OC) from motor vehicles and vegetative burning (39%) (14). The average $PM_{2.5}/PM_{10}$ ratio for the Six Cities Study (3) was 0.6 (based on the 50th percentiles) as compared to a ratio of 0.3 for Phoenix, Arizona (15).

The goal of the present study was to evaluate the associations between daily air pollution and total nonaccidental and cardiovascular mortality in Phoenix. Phoenix is an arid southwestern city with a population of approximately 1 million residents (16). It is an interesting location because of its large proportion of elderly people (9.7% of the population is > 65 years of age). The elderly are more susceptible to air pollution than the general public (2). The primary sources of PM in Phoenix are motor vehicles, paved road dust, and vegetative burning (15).

This study focused on the effects of air pollution on cardiovascular mortality for several reasons. First, the association between air pollutants and cardiovascular mortality has been consistent in previous studies (1,2,9,17). Second, a study in Baltimore, Maryland, found that heart rate variability was associated with $PM_{2.5}$ in elderly subjects with cardiovascular conditions (18). Finally, in this study cardiovascular mortality had the largest sample size, accounting for 45% of the total nonaccidental deaths in the study region (based on zip codes). This may

be reflective of the increased size of Phoenix's elderly population, which is more prone to cardiovascular disease.

A unique aspect of this study is that our pollution data include daily information not only on traditional gaseous pollutants, but also on PM in various size fractions and the chemical composition of $PM_{2.5}$. From 1995 to 1997, the U.S. Environmental Protection Agency (EPA) National Exposure Research Laboratory (NERL) operated a comprehensive monitoring platform in Phoenix. They collected daily $PM_{2.5}$ samples and subsequently analyzed them for various chemical components of PM. This provided an opportunity to examine more specific metrics for PM than simply mass, as well as an opportunity to identify selected chemical components of PM that are associated with mortality.

In addition to PM, this study also evaluated the association between total nonaccidental and cardiovascular mortality and other measured air pollutants: carbon monoxide, nitrogen dioxide, sulfur dioxide, and ozone. These EPA criteria pollutants are also associated with mortality (7,17,19,20).

Methods

Study area and data. Mortality data for all of Maricopa County from 1995 to 1997 were obtained from the Arizona Center for Health Statistics in Phoenix. Death

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We thank D. Bates for his advice and comments regarding the manuscript. We also thank T. Moore for advice regarding the representative spatial scale of the platform particulate matter measurements, C. Mrela for the mortality data, and B. O'Brian for help with assembling the data.

This publication was made possible in part by grant 5T32 ES07262 from the NIEHS, NIH. The U.S. EPA Office of Research and Development partially funded and collaborated in the research described here under assistance agreement R 827355 to the University of Washington.

The contents of this paper are solely the responsibility of the authors and do not necessarily represent the official views of the NIEHS, NIH. This paper has been subjected to EPA review and approved for publication. Mention of trade names or commercial products does not constitute an endorsement or recommendation for use.

Received 8 September 1999; accepted 9 November 1999.

certifi-cate data included residence zip code and the primary cause of death as identified by the *International Classification of Diseases, Ninth Revision (ICD-9)*, World Health Organization, Geneva). Only the deaths of residents in the zip codes located near the air pollution platform were included in this study. This zip-code region was recommended by the Arizona Department of Environmental Quality (Phoenix, AZ). We evaluated total nonaccidental mortality (*ICD-9* codes < 800) and cardiovascular mortality (*ICD-9* codes 390–448.9) in this study. Summary statistics for the mortality outcomes are presented in Table 1.

We obtained $PM_{2.5}$, PM_{10} , PM_{CF} , and $PM_{2.5}$ chemical composition data from the EPA NERL platform in central Phoenix. Chemical composition was only available for $PM_{2.5}$. The monitoring platform is approximately 10 km west-northwest of downtown Phoenix at a state and local air monitoring station. Standard meteorologic parameters such as wind speed and direction, temperature, and relative humidity were continuously measured. The average temperature in Phoenix from 1995 to 1997 was $23.7 \pm 8.1^\circ\text{C}$. The average relative humidity was $32 \pm 15\%$.

NERL investigators made hourly $PM_{2.5}$ and PM_{10} measurements each day using two collocated tapered element oscillation microbalance (TEOM) monitors (Rupprecht & Patasnick Co., Albany, NY). The TEOM- PM_{10} was fitted with an EPA-approved federal reference method PM_{10} impactor inlet (model 246b; Andersen Instruments, Smyrna, GA). The TEOM- $PM_{2.5}$ was fitted with a $PM_{2.5}$ cyclone inlet (University Research Glassware, Chapel Hill, NC). The $PM_{2.5}$ cyclone on the TEOM was replaced with a well-impactor ninety-six (WINS) inlet on 20 December 1996. The WINS inlet has a sharper cut point as compared to the cyclone. We averaged the hourly concentrations to create a 24-hr average (0700–0700 hr), and we calculated the concentration of coarse fraction (TEOM PM_{CF}) as TEOM PM_{10} minus TEOM $PM_{2.5}$.

NERL investigators collected the daily gravimetric integrated 24-hr (starting at 0700) fine particle filter samples using a dual fine particle sequential sampler (DFPSS; University Research Glassware). The DFPSS was fitted with a cyclone that was identical to the cyclone on the TEOM- $PM_{2.5}$. The DFPSS collected daily samples on both Teflon and quartz filters. The Teflon filter was used for mass and elemental analysis, whereas the quartz filter was used for carbon analysis. In addition to the DFPSS, NERL investigators operated a dichotomous sampler (Andersen Instruments, Inc.) every third day beginning 17 June 1996. Both the $PM_{2.5}$ and PM_{CF} samples were collected on Teflon

filters. The investigators measured elemental concentrations at the EPA (Research Triangle Park, NC) with energy dispersive X-ray fluorescence. OC and elemental carbon (EC) were measured by Sunset Laboratory (Forest Grove, OR) using thermal optical transmittance (21).

PM and gaseous pollutant concentrations (range and mean \pm SD) from 1995 to 1997 are presented in Table 2. We obtained gaseous criteria pollutant data for CO, NO_2 , O_3 , and SO_2 from the EPA Aerometric Information Retrieval System (AIRS) database (22) for residential sites in the Phoenix region. We averaged CO values over four monitoring sites and we averaged NO_2 over two sites. Only one residential monitoring site was available for SO_2 . We averaged the hourly averages for CO, NO_2 , and SO_2 over 24 hr from 0700 to 0700. We used the maximum hourly O_3 (O_3 max) concentration in the same 24-hr period in the analysis.

The $PM_{2.5}$ constituents that we evaluated for effects on mortality were sulfur, zinc, lead, soil-corrected potassium (K_S) (23), OC, EC, total carbon (TC), and reconstructed soil. Soil was reconstructed by summing the oxides of Al, Si, Ca, Fe, and Ti using the formula recommended by Malm et al. (24). We also considered $PM_{2.5}$ that was corrected for soil content (nonsoil $PM_{2.5}$ = $PM_{2.5}$ - reconstructed soil). Table 3 presents the percent of the total mass of $PM_{2.5}$ accounted for by each component. The elements aluminum, silicon, calcium, titanium,

and iron were not evaluated separately in the mortality analysis because they are the major elemental components of soil.

Statistical analysis. In our zip-code regions, we analyzed a total of 9,276 nonaccidental deaths from 1995 to 1997. Poisson regression was used to evaluate the association between the air pollutant exposure variables and the mortality outcomes (2,5).

We used Poisson regression because mortality data are discrete counts and death is a rare event. Poisson regression assumes the variance is equal to the mean. When the variance exceeds the mean, the variance is overdispersed. We adjusted standard errors for overdispersion; however, the amount of overdispersion was small. The overdispersion parameter was 1.05 and 1.00 for nonaccidental and cardiovascular mortality, respectively. We calculated all relative risks (RRs) for an interquartile increase (25th–75th percentile) in pollutant concentration.

The effect of air pollution on mortality is small and can be influenced by confounders. Therefore, base models for total mortality and cardiovascular mortality were constructed by adjusting for day of the week with indicator variables, and time trends, temperature, and relative humidity with smoothing functions (25). We determined degrees of freedom (*df*) for the function used to smooth time trend by minimizing autocorrelation as well as the Akaike information criterion (AIC) (26). We chose the *df* and lag for the smoothing functions for temperature and

Table 1. Mortality counts for individuals ≥ 65 years of age in Phoenix.

Year	Total nonaccidental	Average nonaccidental deaths/day	Total cardiovascular	Average cardiovascular deaths/day
1995	3,072	8.45	1,391	3.86
1996	3,201	8.74	1,473	3.98
1997	3,003	8.45	1,318	3.73
1995–1997	9,276	8.55	4,182	3.85

Table 2. Annual range of pollutant concentrations (1995–1997).

Particulate matter pollutant, year	Range	Gaseous pollutant	Range
$PM_{2.5}$ (DFPSS)		CO (ppm)	
1995	4–37		0.5–4.0
1996	3–39		0.3–4.0
1997	2–35		0.3–3.7
3-year mean	12.0 ± 6.6		1.5 ± 0.8
PM_{10} (TEOM)		NO_2 (ppb)	
1995	9–129		8–64
1996	5–213		9–59
1997	7–186		8–61
3-year mean	46.5 ± 22.3		30 ± 10
$PM_{2.5}$ (TEOM)		O_3 max (ppb)	
1995	1–40		10–131
1996	0–42		14–112
1997	1–34		14–104
3-year mean	13.0 ± 7.2		57.0 ± 17.7
PM_{CF} (TEOM)		SO_2 (ppb)	
1995	5–104		0–11
1996	5–187		1–17
1997	5–159		2–12
3-year mean	33.5 ± 17.3		3.1 ± 2.2

relative humidity to minimize the AIC. The base model for total mortality used indicator variables for day of the week, 10 *df* for time trends, 2 *df* for temperature with 2 days lag, and 2 *df* for relative humidity with 0 days lag. The base model for cardiovascular mortality used indicator variables for the day of the week, 10 *df* for time trends, 2 *df* for temperature with 1 day lag, and 2 *df* for relative humidity with 0 days lag.

We included continuous daily data from 1995 to 1997 (1,097 days) in the study. Each day was coded and included in the model to adjust for time trends. Little autocorrelation was observed after adjusting for day of week, time trends, temperature, and relative humidity. The autocorrelation for days 1–25 for both total and cardiovascular mortality were within the 95% confidence interval for an independent series.

We evaluated air pollution exposure variables by adding them individually as linear terms to the base model. The air pollution exposure metrics that were evaluated in this analysis included CO, NO₂, O₃, SO₂, TEOM PM₁₀, TEOM PM_{2.5}, TEOM PM_{CF}, PM_{2.5} (DFPSS), S, Zn, Pb, soil, K_S, nonsoil PM, OC, EC, and TC. Lag days

ranging from 0 to 4 were investigated. We evaluated the assumption of a linear relationship using a smooth function. This assumption was met if a straight line could be placed within the 95% confidence intervals (CIs). A *p*-value < 0.05 associated with the pollution exposure variable was considered significant. We conducted Poisson regression analyses using S-PLUS 4 (Mathsoft, Inc., Seattle, WA)

Factor analysis. We conducted a factor analysis on the daily concentrations of the chemical components of PM_{2.5} from samples collected by the DFPSS (Al, Si, S Ca, Fe, Zn, Mn, Pb, Br, K_S, OC, and EC). The analysis also included the daily averages of the gaseous species emitted by combustion sources (CO, NO₂, and SO₂). Factor analysis is a technique used to explain the correlations between variables in terms of underlying factors that are not directly measurable. Each factor is a linear combination of the original variables and all such factors are orthogonal to each other. The factors were extracted using principal component analysis with a varimax rotation. We conducted factor analysis using SAS (SAS Institute Inc, Cary, NC). We used the resultant factor scores as

surrogate exposure variables in predicting mortality outcomes with the Poisson regression model. Each factor was evaluated in a single source model. However, because the factor scores formed a set of orthogonal variables, we performed a separate regression analysis with all of the scores included in one multifactor model.

We also conducted a factor analysis on the daily concentrations of the chemical components of PM_{CF} from samples collected by the dicot (Al, Si, Cl, S, K Ca, Mn, Fe, Zn, Br, Pb, Sr, Cu, and Rb). We did not use the scores from this analysis in the time-series analysis because the sampling period started in June 1996 and samples were only collected every third day.

Results

Table 4 shows the correlation coefficients between PM, gaseous pollutants, temperature, and relative humidity for Phoenix in 1995–1997. PM_{2.5} (obtained from the DFPSS) was highly correlated with CO (*r* = 0.85) and NO₂ (*r* = 0.79), but less so with SO₂ (*r* = 0.43). PM_{2.5} from the DFPSS was highly correlated with that measured with the TEOM (*r* = 0.93). Table 5 shows the

Table 3. Percent of total mass of PM_{2.5} accounted for by each component.

Component	PM _{2.5} (%)
S ^a	3.69
Mn	0.05
Zn	0.15
Br	0.03
Pb	0.06
OC ^a 1.4	38.37
EC	10.78
K _S	0.52
Soil ^b	17.50

^aIf S is assumed to be in the form of (NH₄)₂SO₄, the mass percent would be 15.2%. ^b2.20% Al + 2.49% Si + 1.63% Ca + 2.42% Fe + 1.94% Ti (23).

Table 4. Correlation coefficients between PM, gaseous pollutants, temperature, and relative humidity (RH) for Phoenix, 1995–1997.

	PM _{2.5} ^a	Temp	PM ₁₀ (TEOM)	RH	PM _{2.5} (TEOM)	PM _{CF} (TEOM)	CO	NO ₂	O _{3max}	SO ₂
PM _{2.5} ^a	1.00	-0.31	0.69	0.16	0.93	0.50	0.85	0.79	-0.24	0.43
Temp	–	1.00	-0.08	-0.55	-0.25	0.00	-0.49	-0.40	0.71	-0.38
PM ₁₀ (TEOM)	–	–	1.00	-0.12	0.77	0.97	0.53	0.53	-0.12	0.41
RH	–	–	–	1.00	0.09	-0.19	0.23	0.08	-0.54	0.10
PM _{2.5} (TEOM)	–	–	–	–	1.00	0.59	0.82	0.77	-0.20	0.48
PM _{CF} (TEOM)	–	–	–	–	–	1.00	0.34	0.37	-0.08	0.33
CO	–	–	–	–	–	–	1.00	0.87	-0.40	0.53
NO ₂	–	–	–	–	–	–	–	1.00	-0.24	0.57
O ₃	–	–	–	–	–	–	–	–	1.00	-0.37
SO ₂	–	–	–	–	–	–	–	–	–	1.00

^aMeasured with the DFPSS.

Table 5. Correlation coefficient matrix of air pollutants.

	S	Zn	Pb	OC	EC	TC	K _S	PM ₁₀	PM _{2.5}	PM _{CF}	Nonsoil PM _{2.5}	Soil	CO	NO ₂	O ₃	O _{3max}	SO ₂
S	1.00	0.14	0.25	0.12	0.04	0.10	0.02	0.19	0.27	0.13	0.26	0.25	0.01	0.04	0.13	0.31	-0.07
Zn	–	1.00	0.63	0.62	0.71	0.65	0.30	0.46	0.61	0.33	0.63	0.49	0.65	0.62	-0.49	-0.27	0.26
Pb	–	–	1.00	0.69	0.69	0.71	0.39	0.48	0.67	0.34	0.71	0.49	0.71	0.63	-0.51	-0.30	0.33
OC	–	–	–	1.00	0.91	0.99	0.65	0.58	0.89	0.38	0.96	0.52	0.89	0.81	-0.57	-0.32	0.49
EC	–	–	–	–	1.00	0.95	0.57	0.58	0.84	0.40	0.89	0.52	0.90	0.82	-0.64	-0.41	0.46
TC	–	–	–	–	–	1.00	0.64	0.59	0.90	0.39	0.96	0.53	0.91	0.83	-0.60	-0.35	0.49
K _S	–	–	–	–	–	–	1.00	0.34	0.59	0.19	0.64	0.26	0.52	0.45	-0.27	-0.14	0.25
PM ₁₀ ^a	–	–	–	–	–	–	–	1.00	0.79	0.97	0.62	0.72	0.55	0.56	-0.25	-0.11	0.42
PM _{2.5} ^a	–	–	–	–	–	–	–	–	1.00	0.60	0.91	0.64	0.82	0.77	-0.44	-0.19	0.47
PM _{CF} ^a	–	–	–	–	–	–	–	–	–	1.00	0.41	0.66	0.37	0.39	-0.14	-0.07	0.35
Nonsoil PM _{2.5}	–	–	–	–	–	–	–	–	–	–	1.00	0.54	0.87	0.80	-0.54	-0.29	0.46
Soil	–	–	–	–	–	–	–	–	–	–	–	1.00	0.48	0.49	-0.17	0.05	0.09
CO	–	–	–	–	–	–	–	–	–	–	–	–	1.00	0.87	-0.68	-0.39	0.51
NO ₂	–	–	–	–	–	–	–	–	–	–	–	–	–	1.00	-0.60	-0.24	0.56
O ₃	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1.00	0.81	-0.46
O _{3max}	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1.00	-0.37
SO ₂	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1.00

^aBased on TEOM measurements.

correlation coefficients between selected chemical composition components of PM_{2.5} and the other air pollutants. TEOM PM₁₀ was correlated with fine soil ($r = 0.72$), OC ($r = 0.58$), EC ($r = 0.58$), and TC ($r = 0.59$). TEOM PM_{2.5} was highly correlated with OC ($r = 0.89$), EC ($r = 0.84$), TC ($r = 0.90$), and to a lesser extent with Zn ($r = 0.61$), Pb ($r = 0.67$), and K_S ($r = 0.59$). The high correlation coefficients between carbon and PM_{2.5} indicate that the majority of the variation in PM_{2.5} is due to combustion products. PM_{CF} was correlated with soil ($r = 0.66$).

OC and EC concentrations follow a seasonal pattern—they are high in the colder months and low in the warmer months. This pattern is due to increased combustion emissions from space heating and the decreased mixing height during the winter months. Particulate sulfur concentrations peak in the warmer months. Soil concentration also follows a seasonal trend, with higher concentrations in the spring and fall. Measured soil concentrations decreased after 20 December 1996 because of the use of the WINS inlet.

Summaries of the RR between the exposure variables and both total and cardiovascular mortality are presented in Tables 6 and 7, respectively. Because of space limitations, we only present statistically significant ($p < 0.05$) and marginally significant ($p < 0.10$) results in the tables, although models were run using all of the pollutants listed in Table 5. Tables of all of the nonsignificant results are available from the authors by request. We evaluated the associations between total and cardiovascular mortality and the gaseous pollutants, PM mass metrics, and PM composition metrics using single-pollutant models.

We found significant associations between both mortality outcomes and selected gaseous air pollutants. CO and NO₂ were positively associated with total mortality at 0- and 1-day lags. There was evidence of a weak association with SO₂ at 0 days lag ($p < 0.10$). We found several strong associations with cardiovascular mortality. Cardiovascular mortality was positively associated with CO (0–4 days lag). This was the most consistent association because the association was significant for all 5 lag days. Statistically significant associations ($p < 0.05$) were also evident with NO₂ on lag days 1 and 4, although the association was weaker on lag days 2 and 3. Cardiovascular mortality was also associated with SO₂ (lag days 2, 3, and 4).

We also found significant associations between the mortality outcomes and particulate mass. The associations between PM₁₀ and total mortality, and between PM_{CF} and total mortality, were marginal ($p < 0.10$). Total mortality was not significantly associated with PM_{2.5}; however, the RR was 1.02 (CI, 1.00–1.05). All PM mass metrics

were associated with an excess risk of cardiovascular death. The strongest associations were with PM_{2.5} (TEOM), followed by PM₁₀ and PM_{CF}. PM_{2.5} adjusted for soil content (nonsoil PM_{2.5}) was also related with cardiovascular mortality with 1 day lag ($p < 0.10$). Table 7 lists all of the statistically significant associations with cardiovascular mortality. Cardiovascular mortality showed a more consistent association with particulate mass concentrations than total mortality. We

further investigated the associations between the mortality outcomes and PM by evaluating the association between the mortality outcomes and the PM composition. The PM_{2.5} composition data analysis revealed that EC and TC were significantly associated with cardiovascular mortality (1 day lag). Weaker associations were also evident with OC at 1 and 3 days lag and TC at 3 days lag. K_S had a significant positive association with cardiovascular mortality (3 day lag).

Table 6. RR for total mortality in Phoenix from an interquartile range (IQR) increase in pollutants.

Pollutant	Lag days	β	SE	t	IQR	RR	LCI	UCI
CO	0	4.50×10^{-2}	1.48×10^{-2}	3.05	1.19	1.06	1.02	1.09
	1	4.15×10^{-2}	1.48×10^{-2}	2.81	1.19	1.05	1.01	1.09
NO ₂	0	2.64×10^0	1.15×10^0	2.31	0.02	1.05	1.01	1.10
	1	3.29×10^0	1.13×10^0	2.91	0.02	1.07	1.02	1.12
	3	1.80×10^0	1.06×10^0	1.69	0.02	1.04	0.99	1.08
	4	2.20×10^0	1.07×10^0	2.05	0.02	1.04	1.00	1.09
SO ₂	0	1.17×10^{-2}	6.37×10^{-3}	1.84	2.78	1.03	1.00	1.07
S	3	-1.38×10^{-4}	6.24×10^{-5}	-2.21	280.60	0.96	0.93	1.00
	4	-1.10×10^{-4}	6.10×10^{-5}	-1.80	279.90	0.97	0.94	1.00
Soil	1	-1.75×10^{-5}	8.67×10^{-6}	-2.01	1,767.45	0.97	0.94	1.00
	2	-1.76×10^{-5}	8.59×10^{-6}	-2.05	1,769.33	0.97	0.94	1.00
	3	-1.75×10^{-5}	8.56×10^{-6}	-2.04	1,772.48	0.97	0.94	1.00
	4	-1.47×10^{-5}	8.54×10^{-6}	-1.72	1,775.62	0.97	0.95	1.00
PM ₁₀ (TEOM)	0	1.06×10^{-3}	5.35×10^{-4}	1.98	24.88	1.03	1.00	1.05
PM _{CF} (TEOM)	0	1.17×10^{-3}	6.99×10^{-4}	1.68	18.39	1.02	1.00	1.05
Pb	3	-2.70×10^{-3}	1.59×10^{-3}	-1.69	6.00	0.98	0.97	1.00

Abbreviations: β , regression coefficient; LCI, lower 95% confidence interval; t , t -statistic from the regression model; UCI, upper 95% confidence interval.

Table 7. RR for cardiovascular mortality from an interquartile range (IQR) increase in pollutants.

Pollutant	Lag days	β	SE	t	IQR	RR	LCI	UCI
CO	0	4.49×10^{-2}	2.14×10^{-2}	2.10	1.19	1.05	1.00	1.11
	1	7.66×10^{-2}	2.07×10^{-2}	3.71	1.19	1.10	1.04	1.15
	2	5.79×10^{-2}	2.00×10^{-2}	2.89	1.19	1.07	1.02	1.12
	3	5.32×10^{-2}	2.03×10^{-2}	2.63	1.19	1.07	1.02	1.12
	4	6.43×10^{-2}	2.06×10^{-2}	3.12	1.19	1.08	1.03	1.13
NO ₂	1	4.88×10^0	1.59×10^0	3.08	0.02	1.10	1.04	1.17
	2	2.53×10^0	1.54×10^0	1.64	0.02	1.05	0.99	1.12
	3	2.76×10^0	1.55×10^0	1.78	0.02	1.06	0.99	1.12
	4	5.74×10^0	1.57×10^0	3.66	0.02	1.12	1.05	1.19
SO ₂	2	1.63×10^{-2}	8.64×10^{-3}	1.88	2.78	1.05	1.00	1.10
	3	1.85×10^{-2}	8.65×10^{-3}	2.14	2.79	1.05	1.00	1.10
	4	2.49×10^{-2}	8.58×10^{-3}	2.90	2.79	1.07	1.02	1.12
K _S	3	5.81×10^{-4}	2.96×10^{-4}	1.97	55.62	1.03	1.00	1.07
PM ₁₀ (TEOM)	0	1.88×10^{-3}	7.66×10^{-4}	2.46	24.88	1.05	1.01	1.09
	1	1.47×10^{-3}	7.56×10^{-4}	1.95	24.88	1.04	1.00	1.08
PM _{2.5} (TEOM)	0	3.91×10^{-3}	2.38×10^{-3}	1.64	8.52	1.03	0.99	1.08
	1	6.85×10^{-3}	2.36×10^{-3}	2.90	8.52	1.06	1.02	1.10
	3	4.86×10^{-3}	2.35×10^{-3}	2.07	8.51	1.04	1.00	1.08
	4	5.43×10^{-3}	2.35×10^{-3}	2.31	8.47	1.05	1.01	1.09
PM _{CF}	0	2.50×10^{-3}	9.88×10^{-4}	2.54	18.39	1.05	1.01	1.09
	1	1.62×10^{-3}	9.78×10^{-4}	1.66	18.39	1.03	0.99	1.07
Nonsoil PM _{2.5}	1	5.56×10^{-6}	3.12×10^{-6}	1.78	6,601.06	1.04	1.00	1.08
OC	1	1.46×10^{-5}	6.82×10^{-6}	2.15	2,976.50	1.04	1.00	1.09
	3	1.39×10^{-5}	6.89×10^{-6}	2.02	2,960.00	1.04	1.00	1.08
EC	1	4.40×10^{-5}	1.82×10^{-5}	2.42	1,165.50	1.05	1.01	1.10
TC	1	1.15×10^{-5}	5.05×10^{-6}	2.28	4,169.00	1.05	1.01	1.09
	3	9.71×10^{-6}	5.10×10^{-6}	1.90	4,170.00	1.04	1.00	1.09

Abbreviations: β , regression coefficient; LCI, lower 95% confidence interval; t , t -statistic from the regression model; UCI, upper 95% confidence interval.

We also found that soil, S, and Pb were negatively associated with total mortality. That is, these exposure variables were associated with a decrease in excess deaths.

We further evaluated the associations between the mortality outcomes and sources of both particulate and gas-phase pollutants using the scores from a factor analysis in place of the individual pollutant concentrations. The results from the analysis with five factors are presented in Table 8. Factor 1 probably represents the influence of motor vehicle exhaust and resuspended road dust with high loadings (loading > 0.5) on Mn, Fe, Zn, Pb, OC, EC, CO, and NO₂. Factor 2 represents soil with high loadings on Al, Si, and Fe. Factor 3 represents vegetative burning with high loadings on OC and K_s. Factor 4 represents a local source of SO₂ with a high loading on SO₂. Factor 5 represents predominantly regional sulfate with a high loading on S. The RRs associated with an interquartile range increase in each factor are presented in Table 9. Total mortality had both a positive and a negative association with the factor representing regional sulfate, positive on lag day 0 (same day) and negative on lag day 3. The factor representing SO₂ had a negative

association with total mortality. We also found a significant negative association for fine soil on lag days 1 and 2, and a nearly significant negative association on lag days 3 and 4. Cardiovascular disease was significantly associated with the factors representing motor vehicles (lag day 1) and vegetative burning (lag day 3). Regression analysis with all of the factors included in a multisource model produced similar results.

Table 10 presents the results from the factor analysis on the daily concentrations of the chemical components of PM_{CF} from samples collected by the dichotomous sampler. Factor 1 represents soil with high loadings on Al, Si, K, Ca, Mn, Fe, Sr, and Rb. Factor 2 represents a source of coarse fraction metals with high loadings on Zn, Pb, and Cu. Factor 3 represents a marine influence with a high loading on Cl. These three factors explain 91.8% of the variance in the PM_{CF} data.

Sensitivity analysis. As a sensitivity analysis, we analyzed temperature as a cofactor rather than a confounder. That is, we evaluated the effects of temperature on mortality as an independent variable rather than adjusting for it in the model as a confounding variable. We evaluated the

significance of temperature after adjusting for day of the week, time trends, and relative humidity. For total and cardiovascular mortality, we found that temperature was not associated with excess deaths. Temperature was not correlated with either PM₁₀ ($r = -0.08$) or PM_{2.5} ($r = -0.25$). A second analysis examined the effect of extreme temperatures. If the average daily temperature was greater than or equal to the 95th percentile (35.4°C), we assigned a 1 to the predictor variable; otherwise we assigned a 0. We did not find an association between extreme temperature and total mortality. However, with cardiovascular mortality, extreme temperature was associated with excess deaths at 0 and 2 days lag ($p < 0.1$). To further assess the importance of the high temperature days to our analysis, we evaluated the association between PM_{2.5} and cardiovascular mortality after excluding the days when the temperature was above the 95th percentile. The effect of eliminating the high temperature days was negligible. The RR for cardiovascular mortality associated with PM_{2.5} (1 day lag) including all days was the same as that excluding the hottest days (RR = 1.06; CI, 1.02–1.10).

We also conducted a sensitivity analysis with relative humidity as a cofactor, with the model controlling for time trends and temperature. As a cofactor, relative humidity was not associated with either total mortality or cardiovascular mortality. To further assess the effects of extreme relative humidity, we eliminated the driest days (relative humidity < 25th percentile) from the data. We then found that the coarse fraction was no longer associated with total mortality. The association between cardiovascular mortality and coarse fraction was statistically significant ($p < 0.05$) on the concurrent day, but nonsignificant with 1-day lag.

We also used dew point rather than relative humidity in the base model. Controlling for dew point rather than relative humidity did not alter our results. We obtained similar regression coefficients.

Table 8. Loadings from factor analysis.

	Factor 1	Factor 2	Factor 3	Factor 4	Factor 5
Al	0.14	0.96	0.08	-0.01	0.07
Si	0.19	0.96	0.11	-0.01	0.10
S	0.04	0.15	0.01	-0.03	0.96
Ca	0.26	0.93	0.15	-0.01	0.09
Mn	0.66	0.62	0.05	0.13	0.07
Fe	0.57	0.76	0.19	0.19	0.05
Zn	0.86	0.24	0.03	-0.03	0.03
Br	0.46	0.31	0.59	0.01	0.28
Pb	0.74	0.21	0.25	0.12	0.26
OC	0.66	0.23	0.55	0.33	0.01
EC	0.76	0.25	0.42	0.28	-0.08
K _s	0.20	0.08	0.92	0.08	-0.04
CO	0.76	0.20	0.39	0.35	-0.09
NO ₂	0.69	0.24	0.31	0.45	-0.05
SO ₂	0.24	-0.04	0.09	0.93	-0.02
Percent variance explained by factor	30.5	27.5	13.7	9.7	7.4

Table 9. RR for total and cardiovascular mortality from an interquartile range (IQR) increase in each factor.

Outcome, factor	Lag days	β	SE	t	IQR	RR	LCI	UCI
Total mortality								
Factor 2	1	-0.03	0.01	-2.03	1.26	0.96	0.93	1.00
	2	-0.04	0.01	-2.45	1.26	0.96	0.92	0.99
	3	-0.02	0.01	-1.67	1.26	0.97	0.94	1.01
	4	-0.02	0.01	-1.74	1.26	0.97	0.94	1.00
Factor 4	2	-0.03	0.01	-2.01	1.09	0.97	0.94	1.00
	4	-0.03	0.01	-1.72	1.09	0.97	0.94	1.00
Factor 5	0	0.03	0.01	2.23	1.38	1.04	1.01	1.08
	3	-0.03	0.01	-2.22	1.39	0.96	0.92	0.99
Cardiovascular mortality								
Factor 1	1	0.05	0.02	2.59	1.11	1.06	1.01	1.10
Factor 3	3	0.05	0.02	2.67	1.02	1.05	1.01	1.09
Factor 5	0	0.04	0.02	2.03	1.38	1.06	1.00	1.12

Abbreviations: β, regression coefficient; LCI, lower 95% confidence interval; t, t-statistic from the regression model; UCI, upper 95% confidence interval.

Table 10. Factor analysis results for PM_{CF}.

Element	Factor 1	Factor 2	Factor 3
Al	0.91	0.33	0.22
Si	0.90	0.36	0.24
Cl	0.25	-0.35	0.82
S	0.59	0.55	0.41
K	0.91	0.33	0.23
Ca	0.84	0.41	0.31
Mn	0.88	0.42	0.17
Fe	0.84	0.50	0.19
Zn	0.47	0.83	0.07
Br	0.23	0.30	0.85
Pb	0.40	0.80	-0.02
Sr	0.83	0.42	0.28
Cu	0.41	0.82	-0.02
Rb	0.91	0.27	0.17
Percent variance explained by factor	51.1	26.5	14.2

To assess the effect of replacing the PM_{2.5} cyclone on the TEOM with the WINS, we evaluated the association between soil and total mortality from 1 January 1995 to 31 December 1996 and from 1 January 1997 to 31 December 1997. The latter period represented the WINS inlet measurements. The association between soil and mortality was not significant for the cyclone measurements alone. Analysis with only the WINS data revealed that the association between soil and mortality was positive and significant at 0 days lag, but not significant for any of the other days.

We estimated soil-related potassium using a correction ratio = K/Si (23). We then reevaluated the RR for cardiovascular mortality associated with K_s using K_s calculated from three slightly different values of K/Si. This correction ratio is dependent on where the soil was obtained: PM_{2.5} paved road dust (K/Si = 1.85/13.69), an agricultural field (K/Si = 1.98/14.35), or Phoenix desert soil (K/Si = 1.89/14.00) (27). We found similar RRs for cardiovascular mortality associated with K_s when we used any of these three approaches. In contrast, total potassium was not associated with either total or cardiovascular mortality.

Discussion

To our knowledge this is the first time-series analysis that has looked at the association between PM chemical composition and mortality and the association between the underlying factors influencing that composition and mortality. Ozkaynak and Thurston (28) combined source apportionment and epidemiologic methods to assess the effects of air pollution on mortality. However, their study was a cross-sectional analysis rather than a time-series analysis. The present study found significant associations between air pollutants and total nonaccidental and cardiovascular mortality. The association between PM₁₀ and cardiovascular mortality is consistent with previous studies. Zmirou et al. (17) reported an RR for cardiovascular mortality from a 50- $\mu\text{g}/\text{m}^3$ increase in PM₁₀ (RR = 1.04) in a study of air pollution in 10 large Western European cities. Pope et al. (5) found an association between respiratory disease death and cardiovascular deaths with PM₁₀ in Utah. Schwartz (2) also found that on high-pollution days (increased total suspended particulates) there was an increased risk of death from cardiovascular disease (RR = 1.09) in Philadelphia, Pennsylvania, and Birmingham, Alabama (1). Furthermore, Anderson et al. (29) found that black smoke was associated with a 0.58% increase in cardiovascular deaths in London.

The association between PM_{2.5} and cardiovascular mortality is similar to that of Schwartz et al. (3), who found that a

10- $\mu\text{g}/\text{m}^3$ increase in PM_{2.5} was associated with a 1.5% increase in total mortality and 2.1% increase in mortality from ischemic heart disease in a study of six eastern U.S. cities. In contrast to Schwartz et al. (3), the present study also found a significant association between PM_{CF} and total and cardiovascular mortality. Although Schwartz et al. (3) did not find a significant association between coarse fraction and mortality when the results from all six cities were combined, there was an association in Steubenville, Ohio, alone. Such observed differences may have been due to differences in regional coarse fraction composition. In Spokane, Schwartz et al. (12) also found no association between coarse particle concentration and total mortality. However, that study only looked at high episodes of coarse particle concentrations resulting from dust storms. Our findings are in agreement with Ostro et al. (13), who found a significant association between daily PM₁₀ dominated by coarse particles and mortality.

We investigated the possibility that PM_{CF} was a surrogate for dryness by eliminating the days with humidity less than the 25th percentile. Although the association with total mortality was no longer significant, we found a significant association with cardiovascular mortality.

The reason for the negative association between soil and total mortality is unclear. One possible explanation for this observation is related to the fact that the PM_{2.5} cyclone on the DFPSS was replaced with a WINS on 20 December 1996. The sharper cut point reduced the amount of soil intrusion into the PM_{2.5} sample, which could produce soil data that are essentially different between the 1995–1996 period and 1997. To assess this effect, we eliminated all 1997 soil data and reevaluated the RR for total mortality. After removing the WINS data, the association between reconstructed soil and total mortality was not significant. These observations are similar to that of Ozkaynak and Thurston (28), who in a study of the association between U.S. mortality rates and particle pollution levels in 1980 found that soil was the least significant predictor of mortality. We also evaluated the association between soil and mortality with only soil data obtained with the WINS. The association was positive and significant ($p < 0.05$) on the concurrent day, but not significant on any other lag days. However, this observation may be due to the low number of days used to evaluate the association between WINS PM_{2.5} soil and total mortality ($n = 377$).

With respect to the elemental components of PM_{2.5}, we found that EC was significantly associated with cardiovascular

mortality. EC is found in combustion-derived particles, most notably diesel exhaust (21). We found that K_s [potassium from vegetative burning (23)] was also associated with cardiovascular mortality.

We found several associations that are potentially spurious. The associations with these variables were found with only total mortality and not with cardiovascular mortality. Lead was negatively associated with total mortality at lag day 3, although this may be reflective of the moderate correlation between Pb and soil in Phoenix ($r = 0.49$). Pb may have accumulated in the soil or in road dust from the past use of leaded gasoline. S was also negatively associated with total mortality on lag day 3. At present, the reason for the negative association with S is unclear. However, S accounts for a relatively small percentage of the mass of PM_{2.5} (15%). The significant negative associations between total mortality and Pb and S were not consistent with the lack of association between these exposure variables and cardiovascular mortality.

For the gaseous species, we found that total nonaccidental mortality and cardiovascular mortality were strongly associated with CO and NO₂. These observations are similar to those of Burnett et al. (30), who found associations between CO and NO₂ and total nonaccidental mortality in Toronto, Canada. Burnett et al. (30) also found that cardiac mortality was associated with CO. CO exacerbates cardiac conditions (10). CO concentrations are also associated with hospital admissions for cardiovascular disease (31). In Phoenix the primary sources of CO and NO₂ are motor vehicles.

The association between SO₂ and cardiovascular mortality was similar to that of Zmirou et al. (17), who also found that an increase in SO₂ was associated with an increase in cardiovascular deaths (RR = 1.04). In addition, Zmirou et al. (17) found weak but significant association between 1-hr maximum O₃ concentrations and cardiovascular mortality (RR = 1.02). Hoek et al. (32) also found an association between total mortality and O₃ in the Netherlands. We found no significant associations with O₃.

The present study demonstrated the use of factor analysis in an epidemiologic study. Using factor analysis, we were able to identify those underlying factors of measured air pollution composition variability that were associated with excess mortality. Poisson regression with factor scores as exposure variables revealed that combustion-related pollutants associated with motor vehicles and vegetative burning as well as fine particulate SO₄ concentrations were significantly associated with cardiovascular mortality. The soil factor, however, was associated with fewer than expected total deaths. These results are

consistent with our time-series results for individual pollutants, specifically CO, NO₂, K_S, EC, OC, and reconstructed soil. It is interesting to note that the factor representing S was significantly associated with cardiovascular mortality, whereas S alone in an individual pollutant model was not associated with cardiovascular mortality. This may be reflective of the contribution of Pb and Br to the S factor.

A unique aspect of this study was the use of the chemical composition data of PM_{2.5}. Using such data, we found positive associations between cardiovascular mortality and K_S, OC, and EC as well as the more traditionally measured pollutants CO, NO₂, SO₂, PM₁₀, PM_{2.5}, and PM_{CF}. Significant associations were also found with factors associated with incomplete combustion products and particulate S compounds. A limitation of this study is that the factor analysis results are only in terms of the variance explained by each factor, rather than in terms of the quantitative contribution from a specific source category. Although methods are available to include quantitative source apportionments in a time-series framework (33), such an analysis is beyond the scope of this initial investigation.

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