The Effects of Components of Fine Particulate Air Pollution on Mortality in California: Results from CALFINE

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OBJECTIVE: Several epidemiologic studies provide evidence of an association between daily mortality and particulate matter < 2.5 µm in diameter (PM2.5). Little is known, however, about the relative effects of PM2.5 constituents. We examined associations between 19 PM2.5 components and daily mortality in six California counties.

DESIGN: We obtained daily data from 2000 to 2003 on mortality and PM2.5 mass and components, including elemental and organic carbon (EC and OC), nitrates, sulfates, and various metals. We examined associations of PM2.5 and its constituents with daily counts of several mortality categories: all-cause, cardiovascular, respiratory, and mortality age > 65 years. Poisson regressions incorporating natural splines were used to control for time-varying covariates. Effect estimates were determined for each component in each county and then combined using a random-effects model.

RESULTS: PM2.5 mass and several constituents were associated with multiple mortality categories, especially cardiovascular deaths. For example, for a 3-day lag, the latter increased by 1.6, 2.1, 1.6, and 1.5% for PM2.5, EC, OC, and nitrates based on interquartile ranges of 14.6, 0.8, 4.6, and 5.5 µg/m³, respectively. Stronger associations were observed between mortality and additional pollutants, including sulfates and several metals, during the cool season.

CONCLUSION: This multicity analysis adds to the growing body of evidence linking PM2.5 with mortality and indicates that excess risks may vary among specific PM2.5 components. Therefore, the use of regression coefficients based on PM2.5 mass may underestimate associations with some PM2.5 components.

KEY WORDS: EC, fine particles, mortality, nitrates, OC, particulate matter, PM2.5, species. Environ Health Perspect 114:13–19 (2007). doi:10.1289/ehp.9281 available via http://dx.doi.org/ [Online 29 August 2006]

Data and Methods

Mortality data. We obtained data on daily mortality for all California residents from the California Department of Health Services, Center for Health Statistics (CDHS), for the period for which data on PM2.5 components were collected: 1 January 2000 through 31 December 2003 (CDHS 1999–2003). We also collected mortality data from 1999 to support additional analyses of PM2.5 (CDHS 1999). A death was included only when it occurred in the decedent’s county of residence. Daily counts of total deaths (minus accidents and homicides) were aggregated for all ages. In addition, we determined daily total mortality counts for those > 65 years of age and for deaths from respiratory disease [International Classification of Diseases, 10th Revision (ICD10; World Health Organization 1993) codes J00–J98] and cardiovascular disease (codes I00–I99).

Pollutant and meteorologic data. We obtained PM2.5 speciation data for the 4-year period 2000 through 2003 from the CARB (CARB 2004). The speciation monitors were part of the State and Local Air Monitoring Stations network, and were filter-based Met One Speciation Air Sampling Systems (Met One Instruments Inc., Grants Pass, OR). We included only counties with ≥ 180 days of observations with PM2.5 species data to ensure sufficient statistical power. Thus, our study of PM2.5 components was limited to deaths occurring in six California counties, which included approximately 8.7 million people, or 25% of the state’s population. Each of the six counties had two monitors measuring PM2.5 components and mass. In three counties (Fresno, Kern, and Riverside), the two monitors were located within four meters of each other.
other in the cities of Fresno, Bakersfield, and Rubidoux, respectively. In the other counties (Sacramento, San Diego, and Santa Clara) the monitors were not co-located. Fresno, Kern, Riverside, and Sacramento Counties reported data every third day, whereas San Diego and Santa Clara Counties reported data every sixth day. For the speciation analyses, the number of observation days available ranged from 243 (San Diego County) to 395 (Sacramento County). The following constituents of PM$_{2.5}$ were measured as 24-hr averages: EC, OC, nitrates (NO$_3^-$), sulfates (SO$_4^{2-}$), aluminum, bromine, calcium, chlorine, copper, iron, potassium, manganese, nickel, lead, sulfur, silicon, titanium, vanadium, and zinc. These PM$_{2.5}$ components represent multiple sources of PM$_{2.5}$, including gasoline combustion, diesel exhaust, wood smoke, crustal material, and secondary pollutants, among others.

We also analyzed PM$_{2.5}$ mass using a larger data set from 1999 through 2003 using all available monitors (including those that did not collect species data) for nine California counties—the same six counties as above plus Contra Costa, Los Angeles, and Orange Counties. The nonspeciated network data were obtained from the CARB (2004). PM$_{2.5}$ monitors were filter-based samplers (model RAA2.5-300; Thermo Andersen, Smyrna, GA). From the nonspeciated network, six counties had only one monitor each collecting daily PM$_{2.5}$ data, whereas Los Angeles, San Diego, and Santa Clara Counties had three, three, and two monitors, respectively.

To allow adjustment for the effect of weather on mortality, we collected daily average temperature and humidity data at meteorologic stations in each of the counties. Hourly temperature data were obtained from the National Oceanic and Atmospheric Administration (NOAA) National Climatic Data Center (NCDC 2004). All daily mortality, pollutant, and meteorologic data were converted into a SAS database (SAS Institute Inc., Cary, NC) and merged by date.

**Methods.** Counts of daily mortality are non-negative discrete integers representing rare events; such data typically follow a Poisson distribution. Therefore, we used Poisson regression, conditional on the explanatory variables. In the basic analytic approach, we used similar model specifications for each city, including smooths for time trend and weather using natural splines. The natural spline model is a generalization of linear regression models for time trend and weather using unlagged values for these covariates, as opposed to the 1-day lag used in the basic model. Third, we examined the effect of alternative specifications of temperature and humidity, using unlagged values for these covariates, as opposed to the 1-day lag used in the basic model. Finally, we stratified the data set by season (April–September) and cool (October–March) periods to examine potential seasonal influences.

All final results were calculated using R (version 2.1.1; R Development Core Team 2004) for the single-county analyses and Stata (StataCorp 2003) for the meta-analyses. To compare relative impacts based on observed concentrations, the results are presented as the excess risk [i.e., (RR-1) × 100] in daily mortality for the interquartile range (IQR) of the pollutants. The full set of results, including the percent change in mortality per microgram per cubic meter for each component, is available online in the Supplemental Material (http://www.ehponline.org/docs/2006/9281/supp.pdf).

**Results**

Table 1 provides descriptive statistics for mortality categories, air quality, and meteorologic data, by county, 2000–2003.

| Mortality category | Contra Costa | Fresno | Kern | Los Angeles | Orange | Riverside | Sacramento | San Diego | Santa Clara |
|--------------------|-------------|--------|------|-------------|--------|-----------|------------|-----------|------------|
| All causes         | 15.7        | 13.4   | 11.4 | 146.7       | 40.2   | 28.8      | 22.0       | 49.5      | 21.3       |
| Cardiovascular disease | 6.4        | 5.8    | 5.1  | 66.3        | 17.8   | 13.2      | 9.3        | 20.3      | 8.7        |
| Respiratory disease | 1.8        | 1.5    | 1.5  | 15.1        | 4.3    | 3.4       | 2.6        | 5.5       | 2.4        |
| Age > 65 years     | 12.2        | 10.1   | 8.4  | 108.6       | 31.8   | 22.8      | 16.3       | 38.8      | 16.4       |

| County characteristics |
|------------------------|
| Population (1,000s)   | 949        | 799    | 662   | 9,519       | 2,846  | 1,545     | 1,223      | 2,814      | 1,683      |
| Mean PM$_{2.5}$ [$\mu$g/m$^3$] | 12.8     | 17.5   | 19.5  | 20.8        | 21.5   | 21.7      | 21.2       | 12.6       | 15.3       |
| Mean temperature ($^\circ$F) | 60.1     | 64.2   | 65.7  | 63.8        | 63.6   | 65.5      | 61.8       | 61.9       | 59.5       |
| Mean relative humidity | 64.4      | 56.5   | 58.2  | 58.8        | 71.8   | 62.6      | 66.1       | 75.8       | 68.3       |
| Days in species analysis | 0        | 355    | 281   | 0           | 0      | 279       | 395        | 243        | 317         |
data from six counties with species data, as well as the other three counties included in the analysis of PM$_{2.5}$ mass concentrations only. Mean daily mortality varied from 147 in Los Angeles County to 11 in Kern County. Mean daily PM$_{2.5}$ concentrations over the study period averaged 19 µg/m$^3$, and ranged from 13 µg/m$^3$ in Sacramento and Contra Costa Counties to 27 µg/m$^3$ in Riverside County, exceeding the U.S. EPA annual average PM$_{2.5}$ standard of 15 µg/m$^3$ in six counties, and the California annual average standard of 12 µg/m$^3$ in all nine counties. Table 2 summarizes the data on PM$_{2.5}$ and its components for the full study period and for the cooler seasons (October–March). Over the four years, there were a total of approximately 1,870 observations across the six counties for most of the species. The largest contributors to PM$_{2.5}$ were EC (5%), OC (37%), NO$_3$ (28%), and SO$_4$ (10%). Table 3 provides the correlations among the species and PM$_{2.5}$. Moderate to high correlations ($r = 0.4–0.6$) were found between PM$_{2.5}$ and EC, OC, NO$_3$, Br, K, and Zn. More modest correlations ($r = 0.2–0.4$) were observed between PM$_{2.5}$ and SO$_4$, Ca, Cu, Fe, Pb, S, Ti, and V. Table 4 provides a summary of the basic meta-analytic results for alternative single-day lags of pollutant concentrations. The results suggest many associations between the pollutants and the mortality end points. Among the pollutants from the speciation network, the strongest associations were observed for PM$_{2.5}$ mass, EC, NO$_3$, Cl, Cu, Fe, K, Ti, V, and Zn. Adding observations to PM$_{2.5}$ mass by using data from the nonspeciation counties (so that all nine counties were included) enhanced the statistical power and resulted in observable associations with all four of the mortality categories. When the results by mortality end points were examined, several patterns emerged. All-cause mortality was associated most strongly with Cu and PM$_{2.5}$ext, with weaker associations also observed with NO$_3$ and Cl. Cardiovascular mortality was associated most strongly with EC, K, Zn and PM$_{2.5}$ext with more modest associations observed with OC, NO$_3$, Fe, and Ti. Respiratory mortality was associated with Cu and Ti, with weaker associations with V, Zn, and PM$_{2.5}$ext. Finally, for mortality among those > 65 years of age, significant associations were observed with PM$_{2.5}$ext, NO$_3$, Cl, K, and Zn.

Figure 1 summarizes the quantitative meta-analytic results for all-cause and cardiovascular mortality using single-day lags of selected pollutants (the full set of results is available in the Supplemental Material: http://www.ehponline.org/docs/2006/9281/suppl.pdf ). Unlike many time-series studies with continuous daily data, not all lags refer to the same outcome days. Specifically, for PM data collected every third day, lags 0 and 3 will generally refer to the same days (and numbers of deaths per day) except at the ends of the time series. However, for those same PM data, lags 1 and 2 refer to different days with different numbers of deaths. Although this phenomenon holds true for other studies using nondaily PM data, the number of observations used in this analysis is small relative to those in most published studies of PM and mortality. Therefore, the results...
are somewhat sensitive to the specified lag; however, the findings suggest many associations between the pollutants and mortality endpoints. For example, for a 3-day lag, cardiovascular mortality increased by 1.6% (95% confidence interval (CI), 0.3–3.9) for EC, 1.6% (95% CI, 0.3–3.9) for EC, 1.5% (95% CI, –0.2 to 3.3) for nitrates and 2.2% (95% CI, 0.3–4.2) for Zn for IQRs of 14.6, 0.8, 4.6, 5.5, and 0.01 μg/m³, respectively. Most CIs are large due to the relatively low numbers of observations. In comparing the beta coefficients, the percent change in cardiovascular mortality per microgram per cubic meter was much greater for many of the components relative to PM2.5 mass (see Supplemental Material: http://www.ehponline.org/docs/2006/9281/suppl.pdf).

Table 5 and Figure 2 summarize the cool season–specific results. During the cooler months, there are more associations between the pollutants and mortality than when the entire year is included in the analysis. Except for Al, Br, and Ni, almost all of the pollutants were associated with all-cause and cardiovascular mortality, and with daily deaths among those > 65 years of age. In contrast, during the summer months there were few associations, except for K with cardiovascular and respiratory deaths, and Al, Cl, Cu, Pb, Ti, and Zn with respiratory mortality (data not shown). Additional sensitivity analyses indicated that the species results were insensitive to treatment of missing values, alternative df used for the smoothers of time and weather, and different lags for the weather terms in the model specifications (data not shown).

Discussion
In this time-series analysis of PM in California, ambient concentrations of several constituents of fine particles were associated with daily mortality. Specifically, the data suggest consistent associations with EC, OC, NO₃, Cu, K, Ti, and Zn, as well as with PM₂.₅ mass. Stronger associations were observed with mortality for cardiovascular disease and among those > 65 years of age. For cardiovascular mortality, risks associated with the IQRs of EC and Zn were particularly elevated. Comparison of the pollution regression coefficients indicated that, in general, EC and many of the other species that contribute significantly to PM₂.₅ mass, including OC, NO₃, and Zn, all demonstrated higher excess risks than PM₂.₅ mass. Although this observation may be partly the result of stochastic variability, the associations with mortality were all the more striking given the relatively small number of days with species data in each county (range 243–395), because most time-series studies have > 1,000 days of

Table 4. Summary of statistically significant associations between mortality and alternative pollutant lags (numbers in the table indicate whether single lags of 0–3 days were statistically significant).

| Species and lag day | All-cause | Cardiovascular | Respiratory | Age > 65 years |
|---------------------|-----------|----------------|-------------|---------------|
| PM₂.₅c | — | 1*, 3* | — | 3** |
| EC | — | 3** | — | — |
| OC | — | 3* | — | — |
| NO₃ | 0* | 3* | — | 0** |
| SO₂ | — | — | — | — |
| Al | — | — | — | — |
| Br | — | — | — | — |
| Ca | — | — | — | — |
| Cl | — | — | 3* | — |
| Cu | 1** | — | — | — |
| Fe | — | 2* | — | — |
| K | — | 2** | — | 2* |
| Mn | — | — | — | — |
| Ni | — | — | — | — |
| Pb | — | — | — | — |
| S | — | — | — | — |
| Si | — | — | — | — |
| Ti | — | — | — | — |
| V | — | 1* | — | — |
| Zn | 3** | — | 1* | 1**, 3** |
| PM₂.₅extb | 0**, 1* | 0**, 1** | 1*, 2* | 0**, 1** |

The regression model includes time (4 df/year), 1-day lags of temperature and humidity (3 df), day of week, and pollutant.

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*Includes six counties with species data, 2000–2003. *Includes all nine counties in Table 1, 1999–2003. *p < 0.10; **p < 0.05.

Figure 1. Excess risk [mean (95% CI)] of mortality per IQR of concentrations. (A) All-cause mortality. (B) Cardiovascular mortality.

* p < 0.10. ** p < 0.05.
data (HEI 2003). Increasing the sample size increased the strength of the PM$_{2.5}$ associations with mortality. With few exceptions, these results were relatively insensitive to alternative treatment of missing values, different smoothers of time, and different lag specifications for meteorologic covariates. Results were somewhat sensitive, however, to the lag day examined. Most of the associations were with a 1-day lag, which is fairly consistent with many previous time-series studies of PM $<$ 10 μm in aerodynamic diameter (PM$_{10}$) and PM$_{2.5}$ (HEI 2003). Although there is increasing evidence linking PM exposures with cardiovascular pathophysiology (Brook et al. 2004), there is little to justify a priori an appropriate lag structure for the vast majority of PM$_{2.5}$ constituents. In this analysis, it is unclear whether the associations of mortality with different lags were caused by a) different mechanisms; b) different mortality reference days for lags 1 and 2 versus lags 0 and 3 because the exposure data were not collected on a daily basis (see “Methods”); or c) stochastic variability due to the relatively low number of observations.

We found stronger and more frequent associations between mortality and PM$_{2.5}$ components during the cooler months, when most (but not all) components have higher concentrations. For example, the warm and cool season averages for PM$_{2.5}$ were 14 and 24.6 μg/m$^3$, respectively. For EC, OC, and NO$_3$, the cool season averages were roughly twice those of the warm season. These differences represent seasonal variation in sources (e.g., residential wood combustion), particle chemistry and meteorology. For example, Lipsett et al. (1997) reported that during the winter in Santa Clara County, residential wood combustion accounts for as much as 45% of PM$_{10}$. Moreover, in the winter months the inversion layers and vertical mixing depths throughout much of the state tend to be much shallower than in warmer months. In addition, the generally mild climate in California in the cooler months may mean that more windows are open, resulting in greater indoor penetration of outdoor pollutants relative to the summer months when air conditioner use is more common.

Becker et al. (2005) reported seasonal variation in the toxicity of PM, based on in vitro analysis of markers of inflammation and oxidative stress, which they hypothesized could be explained by temporal differences in particle composition. In our analysis, evidence of seasonally different effects for a specific PM$_{2.5}$ species suggests that differences in both composition and exposure patterns may be important. Our findings differ somewhat with the time-series mortality analysis of PM$_{10}$ in 100 U.S. cities by Peng et al. (2005). That study reported stronger effects during the summer months, based on observations from October–March.

### Table 5. Summary of statistically significant associations between mortality and alternative pollutant lags during October–March, 2000–2003 (numbers in the table indicate whether single lags of 0–3 days were statistically significant).

| Components | All-cause | Cardiovascular | Respiratory | Age $>$ 65 years |
|------------|-----------|----------------|-------------|-----------------|
| PM$_{2.5}$ | 3**       | 3**            | —           | 3**             |
| EC         | 3**       | —              | —           | 3               |
| OC         | 3**       | —              | —           | 3**             |
| NO$_3$     | 0**, 3**  | 0*, 3**        | —           | 0**             |
| SO$_4$     | 0**, 3**  | 0*, 3**        | —           | 0**             |
| PM$_{2.5ext}$ | 0**, 1**, 2**, 3** | 0**, 1**, 2**, 3** | 0**, 1**, 3** | 0**, 1**, 3**, 3** |

The regression model includes time (4 df/year), 1-day lags of temperature and humidity (3 df), day of week, and pollutant. *p < 0.10; **p < 0.05.

### Figure 2. Excess risk [mean (95% CI)] of mortality per IQR of concentrations for the cooler months (October–March). (A) All-cause mortality. (B) Cardiovascular mortality.

* $p < 0.10; **p < 0.05.$
cities primarily in the Northeast. Their base case region-specific analysis showed a modest warm season effect for the Northwest (which included Northern California) but no season-specific effect for Southern California, the only region that did not show a larger effect in summer in their analysis. However, the latter results appear to be sensitive to the df in the smooth of time and the PM$_2.5$ lag used. For example, if 3 or 5 df for time smooth or an unlagged PM$_{10}$ was specified, the effects were larger in the nonsummer months, a result consistent with our findings. In comparison, our results were not affected by use of alternative df for the smooth of time, but were sensitive to the specified lag of pollution.

A few previous studies have examined the associations between some species of PM and daily mortality. For example, Fairley (2003) examined the impacts of NO$_x$, SO$_x$, and coefficient of haze (COH) in Santa Clara County. The latter is highly correlated with EC, and is likely to be a good marker of particulate pollution from motor vehicles, especially diesel exhaust, and from wood smoke. All three PM$_{2.5}$ constituents were associated with all-cause mortality, whereas NO$_x$ was also associated with cardiovascular mortality. These findings were consistent with those of Hoek (2003) in the Netherlands, where associations with mortality were reported for SO$_x$, NO$_x$, and black smoke. In a study in Buffalo, New York, Gwynn et al. (2000) reported associations of COH, SO$_x$, and hydrogen ion (a measure of aerosol acidity) with total mortality. Ito (2003) failed to find associations of mortality with SO$_x$ or hydrogen ion in Detroit, Michigan, although only limited data for these pollutants were available. In their study of the eight largest Canadian cities, Burnett et al. (2000) examined the impact of 47 separate constituents of PM$_{2.5}$. Within the fine fraction, SO$_x$, Zn, Ni, and Fe were all associated with mortality, as was COH. NO$_x$, EC, and OC were not measured in the Canadian study. Mar et al. (2000) reported associations between mortality in Phoenix, Arizona, and EC, OC, and K. Finally, in analyses of emergency department visits, Metzger et al. (2004) reported associations of both EC and OC with visits for cardiovascular mortality, including residential wood combustion (Maykut et al. 2003; Watson et al. 2001).

The use of multiple cities increased the statistical power and reduced the likelihood that these results were due to factors associated with one geographic location. The association with mortality of any single substance, however, may be a result, at least in part, of its own toxicity or of exposures to other substances with which it is highly correlated. In future work, we will examine the impact of specific sources through use of source profiles for the six California counties based on chemical mass balance models (Thurston et al. 2005). It is important to note the limitations of our data. First, the use of a single location for monitoring PM$_{2.5}$ components in several of the counties is likely to lend to random measurement error and the potential for downward-biased effect estimates. Second, because every-day monitoring was not available, we were unable to estimate the impact of cumulative exposures, which tend to generate larger effect estimates than that of a single-day lag (Schwartz 2000). Third, given the numbers of pollutants and end points examined and the relatively low number of observations, it is possible that some of the results may have occurred by chance. Finally, there may be differential measurement error among the components both with respect to spatial variability and indoor/door penetration. For example, Janssen et al. (2005) analyzed the longitudinal correlation of personal and outdoor concentrations of several PM$_{2.5}$ species in Helsinki, Finland, and Amsterdam, the Netherlands.

Our findings add to the growing body of evidence linking PM$_{2.5}$ with mortality and indicate that excess risks may vary with the specific PM$_{2.5}$ constituent. The results also support the hypothesis that pollution from motor vehicles and other sources of combustion, including residential wood burning, may be of particular concern. Finally, the use of regression coefficients based only on PM$_{2.5}$ mass may underestimate the effects of some of its specific components.

REFERENCES

Becker S, Dailey LA, Soukup JM, Grombaw SC, Devlin RB, Huang Y-T. 2005. Seasonal variations in air pollution particle–induced inflammatory mediator release and oxidative stress. Environ Health Perspect 113:1032–1038.

Blanchard C. 2003. Spatial and temporal characterization of particulate matter. In: Particulate Matter Science for Policy Makers: A NABTAE Assessment (McMurry PH, Shepherd MF, Vickery JS, eds). Cambridge, UK:Cambridge University Press, 191–231.

Brook RD, Franklin B, Cacicia W, Hong Y, Howard G, Lipsett M, et al. 2004. Air pollution and cardiovascular disease: a statement for healthcare professionals from the Expert Panel on Population and Prevention Science of the American Heart Association. Circulation 109:2659–2671.

Burnett RT, Brook JR, Dann T, Deloaca C, Philips O, Calmack S, et al. 2000. Association between particulate- and gas-phase components of urban air pollution and daily mortality in eight Canadian cities. Inhal Toxicol 12(suppl 4):15–39.

CARB California Air Resources Board. Air Quality Data Section, Planning and Technical Support Division. Sacramento:California Air Resources Board.

CDHS (California Department of Health Services). 1999–2003. Death Statistical Master Files, 1999–2003. Sacramento, CA:Center for Health Statistics.

Der Simonian R, Laird N. 1986. Meta-analysis in clinical trials. Control Clin Trials 7:177–188.

Fairley D. 2003. Mortality and air pollution for Santa Clara County, California, 1989–1996. In: Revised Analyses of Time-Series Studies of Air Pollution and Health. Special Report. Boston:Health Effects Institute, 97–106. Available: http://www.healtheffects.org/Pubs/TimeSeries.pdf [accessed 16 February 2006].

Gwynn RC, Burnett RT, Thurston GD. 2001. A time-series analysis of acid particulate matter and daily mortality and morbidity in the Buffalo, New York, region. Environ Health Perspect 108:125–133.

HEI. 2003. Revised analyses of the National Morbidity, Mortality, and Air Pollution Study (NMMAPS), Part II. In: Revised Analyses of Time-Series Studies of Air Pollution and Health, Special Report. Boston:Health Effects Institute, 9–72. Available: http://www.healtheffects.org/Pubs/TimeSeries.pdf [accessed 16 February 2006].

Huang Y-CT. 2005. Seasonal variations in air pollution and daily mortality in eight Canadian cities. Inhal Toxicol 12(suppl 4):15–39.

Mar et al. 2000. Air pollution and cardiovascular disease: a statement for healthcare professionals from the Expert Panel on Population and Prevention Science of the American Heart Association. Circulation 109:2659–2671.

Burnett RT, Brook JR, Dann T, Deloaca C, Philips O, Calmack S, et al. 2000. Association between particulate- and gas-phase components of urban air pollution and daily mortality in eight Canadian cities. Inhal Toxicol 12(suppl 4):15–39.

CARB California Air Resources Board. Air Quality Data Section, Planning and Technical Support Division. Sacramento:California Air Resources Board.

CDHS (California Department of Health Services). 1999–2003. Death Statistical Master Files, 1999–2003. Sacramento, CA:Center for Health Statistics.

Der Simonian R, Laird N. 1986. Meta-analysis in clinical trials. Control Clin Trials 7:177–188.

Fairley D. 2003. Mortality and air pollution for Santa Clara County, California, 1989–1996. In: Revised Analyses of Time-Series Studies of Air Pollution and Health. Special Report. Boston:Health Effects Institute, 97–106. Available: http://www.healtheffects.org/Pubs/TimeSeries.pdf [accessed 16 February 2006].

Gwynn RC, Burnett RT, Thurston GD. 2001. A time-series analysis of acid particulate matter and daily mortality and morbidity in the Buffalo, New York, region. Environ Health Perspect 108:125–133.

HEI. 2003. Revised analyses of the National Morbidity, Mortality, and Air Pollution Study (NMMAPS), Part II. In: Revised Analyses of Time-Series Studies of Air Pollution and Health, Special Report. Boston:Health Effects Institute, 9–72. Available: http://www.healtheffects.org/Pubs/TimeSeries.pdf [accessed 16 February 2006].

Hoeck G. 2003. Daily mortality and air pollution in the Netherlands. In: Revised Analyses of Time-Series Studies of Air Pollution and Health. Special Report. Boston:Health Effects Institute, 9–72. Available: http://www.healtheffects.org/Pubs/TimeSeries.pdf [accessed 16 February 2006].

Hoek G. 2003. Daily mortality and air pollution in the Netherlands. In: Revised Analyses of Time-Series Studies of Air Pollution and Health. Special Report. Boston:Health Effects Institute, 133–142. Available: http://www.healtheffects.org/Pubs/TimeSeries.pdf [accessed 2 February 2006].

Ito K. 2003. Associations of particulate matter components with daily mortality and morbidity in Detroit, Michigan. In: Revised Analyses of Time-Series Studies of Air Pollution...
and Health. Special Report. Boston: Health Effects Institute, 143–156. Available: http://www.healtheffects.org/Pubs/TimeSeries.pdf [accessed 16 February 2006].

Janssen NAH, Lanki T, Hoek G, Vallius M, de Hartog JJ, Van Grieken R, et al. 2005. Associations between ambient, personal, and indoor exposure to fine particulate matter constituents in Dutch and Finnish panels of cardiovascular patients. Occup Environ Med 62:866–877.

Janssen NAH, Schwartz J, Zanobetti A, Suh H. 2002. Air conditioning and source-specific particles as modifiers of the effect of PM<sub>2.5</sub> on hospital admissions for heart and lung disease. Environ Health Perspect 101:43–49.

Laden F, Neas LM, Dockery DW, Schwartz J. 2000. Association of fine particulate matter from different sources with daily mortality in six U.S. cities. Environ Health Perspect 108:941–947.

Lewis CW, Norris GA, Conner TL, Henry RC. 2003. Source apportionment of Phoenix PM<sub>2.5</sub> aerosol with the Unmix receptor model. J Air Waste Manag Assoc 53:325–338.

Lipsett M, Hurley S, Ostro B. 1997. Air pollution and emergency room visits for asthma in Santa Clara County, California. Environ Health Perspect 105:216–222.

NCDC (National Climatic Data Center). 2004. Climate Resources. Available: http://www.ncdc.noaa.gov/oa/climate/climatetources.html [accessed 4 April 2005].

NRC (National Research Council). 2004. Research Priorities for Airborne Particulate Matter. IV. Continuing Research Progress. Washington, DC: National Academies Press.

Peng RD, Dominici R, Pastor-Barriuso R, Zeger SL, Samet JM. 2005. Seasonal analysis of air pollution and mortality in 100 U.S. cities. Am J Epidemiol 161:585–594.

R Development Core Team. 2004. R: A Language and Environment for Statistical Computing. Vienna, Austria: R Foundation for Statistical Computing. Available: http://www.R-project.org [accessed 6 September 2005].

Schwartz J. 2000. The distributed lag between air pollution and daily deaths. Epidemiology 11:320–326.

StataCorp. 2003. Stata version 8. College Station, TX: StataCorp.

Thurston GD, Ito K, Mar T, Christensen W, Eatough DJ, Henry RC, et al. 2005. Workgroup report: workshop on source apportionment of particulate matter health effects—intercomparison of results and implications. Environ Health Perspect 113:1768–1774.

Watson JG, Chow JC, Houck JE. 2001. PM<sub>2.5</sub> chemical source profiles for vehicle exhaust, vegetative burning, geological material, and coal burning in Northwestern Colorado during 1995. Chemosphere 43:1141–1151.

World Health Organization. 1993. International Classification of Diseases, 10th Revision. Geneva: World Health Organization.