



## Coarse and fine particles and daily mortality in the Coachella Valley, California: a follow-up study

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Many epidemiological studies provide evidence of an association between ambient particles, measured as PM<sub>10</sub>, and daily mortality. Most of these studies have been conducted in urban areas where PM<sub>10</sub> is highly correlated with and dominated by fine particles less than 2.5  $\mu\text{m}$  in diameter (PM<sub>2.5</sub>). Fewer studies have investigated impacts associated with the fraction of coarse mode particles (between 2.5 and 10  $\mu\text{m}$  in diameter). In a previous study using data from 1989 through 1992 in the Coachella Valley, a desert resort and retirement area east of Los Angeles, we reported associations between PM<sub>10</sub> and several different measures of mortality [Ostro B.D., Hurley S., and Lipsett M.J. Air pollution and daily mortality in the Coachella Valley, California: a study of PM<sub>10</sub> dominated by coarse particles. *Environ. Res.* 1999; 81: 231–238]. In this arid environment, coarse particles of geologic origin are highly correlated with and comprise approximately 60% of PM<sub>10</sub>, increasing to >90% during wind events. This study was intended to repeat the earlier investigation using 10 years (1989–1998) of daily data on mortality and PM<sub>10</sub>. The last 2.5 years of data also included daily measures of PM<sub>2.5</sub>, allowing examination of size-specific impacts. To ensure adequate statistical power, we attempted to develop predictive models for both fine and coarse particles to use in analyses of the full 10-year period. An acceptable fit was found only for coarse particles, which were found to be a cubic function of PM<sub>10</sub> ( $R^2=0.95$ ). Outcome variables included several measures of daily mortality, including all-cause (minus accidents and homicides), cardiovascular and respiratory mortality. Multivariate Poisson regression analyses using generalized additive models were employed to explain the variation in these endpoints, controlling for temperature, humidity, day of the week, season, and time, using locally weighted smoothing techniques. Pollution lags of up to 4 days were examined. Several pollutants were associated with all-cause mortality, including PM<sub>2.5</sub>, carbon monoxide and nitrogen dioxide. More consistent results were found for cardiovascular-specific mortality, for which associations were found for coarse particles (RR=1.02; 95% C.I., 1.01–1.04), PM<sub>10</sub> (RR=1.03; 95% C.I., 1.01–1.05). None of the pollutants was associated with respiratory-specific mortality. Ozone was not associated with any of the mortality outcomes. These findings are generally consistent with those we previously reported for the Coachella Valley for the period 1989–1992, demonstrating associations between several measures of particulate matter and daily mortality in an environment in which particulate concentrations are dominated by the coarse fraction. *Journal of Exposure Analysis and Environmental Epidemiology* (2000) 10, 412–419.

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### Introduction

In most urban areas of the United States, ambient PM<sub>10</sub> (particles less than 10  $\mu\text{m}$  in diameter) is dominated by the fine fraction less than 2.5  $\mu\text{m}$  in diameter (PM<sub>2.5</sub> or fine particles). PM<sub>10</sub> has been repeatedly associated with a wide range of adverse health outcomes, including mortality, and it has been proposed that PM<sub>10</sub>-associated toxicity is attributable principally to the fine fraction (U.S. EPA, 1996). However, the potential impacts of the coarse fraction (PM<sub>10</sub> minus PM<sub>2.5</sub>) have been less well documented. Specifically, there have been few studies examining whether exposures to ambient particulate matter

originating primarily from geologic sources (e.g., soil and sand) rather than combustion are associated with adverse health outcomes.

Coachella Valley is roughly 100 miles east of Los Angeles and includes the cities of Palm Springs at the northwestern end of the populated corridor in the Valley and Indio towards the southern end. The Valley is a desert area with hot summers and mild winters, and is bordered by mountains on the north, east and west and by the Salton Sea on the south. Geologic particles comprise a significant percentage of the annual average particulate mass (approximately 60% of PM<sub>10</sub>), increasing to >90% during wind episodes that occur throughout the year.

In a previous study using data from 1989 through 1992, we reported associations between daily concentrations of PM<sub>10</sub> and several different measures of mortality in the Valley (Ostro et al., 1999). The current study was intended to repeat the earlier investigation using 10 years (1989–1998) of daily data on mortality and PM<sub>10</sub>. In addition,

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during the final 2.5 years in this period, daily data on PM<sub>2.5</sub> were collected.

## Data

Data on daily mortality for the Coachella Valley were obtained from the California Department of Health Services, Health Data and Statistics Branch, for the period January 1, 1989 through December 10, 1998, a total of 3683 days (less 6 days with missing mortality data). Daily counts of total deaths (minus accidents and homicides) were aggregated as well as total daily counts of deaths from respiratory and cardiovascular diseases (International Classification of Diseases, Ninth Revision [ICD-9] codes 460–519 and 393–440, respectively). Because the population of the Coachella Valley varies throughout the year, with a large influx of tourists during the winter and early spring, deaths of individuals listed with a permanent address in zip codes outside the Valley were excluded from the analyses.

Pollutant data were obtained from the South Coast Air Quality Management District, which operates and maintains two fixed-site monitoring stations in the Valley. The monitors in Palm Springs measured ozone, nitrogen dioxide and carbon monoxide every hour, while PM<sub>10</sub> was collected every sixth day. At the Indio monitoring site, situated approximately 25 miles to the Southeast, ozone and PM<sub>10</sub> were monitored continuously throughout the study period. Hourly PM<sub>10</sub> data were collected using a  $\beta$ -attenuation monitor (Graseby Andersen Model FH62I-N). For the gaseous pollutants, both 1-h maxima and longer term averages were computed for each day of the study period. PM<sub>10</sub> was expressed both as a 24-h average and a 1-h maximum. Supplementary data on PM<sub>10</sub> and PM<sub>2.5</sub> were collected from April 1996 through November 1998. The PM<sub>2.5</sub> data were collected at both Palm Springs and Indio using  $\beta$ -attenuation monitors, while an additional continuous monitor for PM<sub>10</sub> was deployed at the Palm Springs site. Since PM<sub>2.5</sub> data were only available for 2.5 years, predictive models were estimated for both PM<sub>2.5</sub> and the coarse particle fraction (CF=PM<sub>10</sub>–PM<sub>2.5</sub>) to obtain measures of these pollutants for the full 10 years. Since the PM<sub>10</sub> data were more complete at Indio ( $n=3011$ ) than at Palm Springs ( $n=1287$ ), we used the former for most of the subsequent analyses.

We used a statistical model to examine both fine and coarse particles as a function of PM<sub>10</sub> (considering linear, quadratic and cubic terms), other pollutants, weather variables, time and season. Ultimately, CF in Indio was estimated, using ordinary least squares, as a cubic function of PM<sub>10</sub> with an  $R^2$  of 0.95. The cubic function generated a slightly better fit than the quadratic, and was of the form  $CF = -1.86 + 0.51 \times PM_{10} + 0.003 \times PM_{10}^2 - (5.94 \times 10^{-6}) \times PM_{10}^3$ . Given this good fit, models were run in the full

analysis using the estimated values for CF. For PM<sub>2.5</sub>, models were estimated for both Palm Springs and Indio. For Indio, the best predictive model had a much lower  $R^2$  (0.37), while a slightly better model was developed for the Palm Springs data ( $R^2=0.53$ ). Given the poor fit, predicted values of PM<sub>2.5</sub> were not used in subsequent analysis; rather, only the 2-1/2 years of measured values of PM<sub>2.5</sub> were used.

To adjust for potential effects of weather on mortality, daily meteorological data collected at airports in Palm Springs and Thermal (located a few miles south of Indio) were obtained from the National Climatic Data Center (Asheville, NC). These data consisted of precipitation, minimum and maximum temperatures, visual range and relative humidity at 2 p.m.

## Methodology

Counts of daily mortality are nonnegative discrete integers representing rare events; such data typically follow a Poisson distribution. Therefore, the principal analysis relied on Poisson regression, conditional on the explanatory variables. Poisson regressions are a subclass of log-linear models in which the natural logarithm of the expected counts is modeled as a linear sum of the explanatory variables, with the distribution of the errors assumed to be Poisson. We calculated relative risk estimates by exponentiating the product of the pollutant-specific regression coefficient and the interquartile range (25th–75th percentiles) for that pollutant.

The model-building strategy for the multivariate analysis involved construction of a basic de-trended model in which variations due to weather conditions and temporal trends in the data were removed. Because the unit of analysis in this study is the day, potential confounders include factors that may affect daily mortality and are likely to vary over time in concert with air pollution levels. To develop our regression model, we determined the best fit of several covariates prior to the entry of air pollution variables into the model. We examined, in turn, the association of each outcome with temperature, humidity and dewpoint (including lags of up to 4 days for each meteorological variable), day of the week, and time.

Visual inspection of the mortality data suggested a modest seasonal pattern. We controlled for seasonality and population growth using a generalized additive model (GAM). We incorporated a locally weighted (loess) smooth of time, which can accommodate nonlinear and nonmonotonic patterns between time and mortality, offering a flexible nonparametric modeling tool (Hastie and Tibshirani, 1990). In addition, a loess smooth of time diminishes short-term trends in the data, thereby helping to reduce the degree of serial correlation.

**Table 1.** Descriptive statistics for Coachella Valley: Jan 1, 1989 through Dec 10, 1998.

Variable	N (# days)	Mean	Min	Max
Total deaths	3677	5.8	0	17
Total cardiovascular deaths	3677	2.7	0	10
Total respiratory deaths	3677	0.52	0	5
PM10 ( $\mu\text{g}/\text{m}^3$ , 24-h avg, Palm Springs)	1287	29.8	6	183
PM10 ( $\mu\text{g}/\text{m}^3$ , 24-h avg, Indio)	3011	47.4	3	417
PM2.5 ( $\mu\text{g}/\text{m}^3$ , 24-h avg, Palm Springs)	1011	12.7	0	165
PM2.5 ( $\mu\text{g}/\text{m}^3$ , 24-h avg, Indio)	1041	16.8	5	48
Coarse particles ( $\mu\text{g}/\text{m}^3$ , 24-h avg, Palm Springs)	990	17.9	0	149
Coarse particles ( $\mu\text{g}/\text{m}^3$ , 24-h avg, Indio)	789	25.8	0	164
Predicted coarse particles ( $\mu\text{g}/\text{m}^3$ , 24-h avg, Indio)	2990	30.5	0	418
Ozone (pphm, 1-h max, Palm Springs)	3558	6.7	0	19
Ozone (pphm, 1-h max, Indio)	3488	6.2	0	18
Ozone (pphm, 8-h avg, Palm Springs)	3558	5.1	0	13
Nitrogen dioxide (pphm, 24-h avg, Palm Springs)	3421	2.0	0	6
Carbon monoxide (ppm, 8-h avg, Palm Springs)	3502	0.3	0	2.25
Maximum daily temperature ( $^{\circ}\text{F}$ )	3462	88.7	49	125

Once the covariates most strongly associated with daily total mortality were determined, each pollutant was added separately to the model. Contemporaneous exposure and lags of up to 4 days were examined to allow for a delayed effect of exposure. In addition, a 4-day moving average exposure (including lags 0 to 3) was examined. In this way, we examined the relationships of daily mortality to ambient concentrations of PM10, PM2.5, CF, ozone, nitrogen dioxide, and carbon monoxide, in both single- and multi-pollutant models.

Several sensitivity analyses were also conducted. First, in addition to the three principal mortality categories (total, cardiovascular, and respiratory), we considered total mortality minus the sum of cardiovascular and respiratory deaths as a “control” outcome (i.e., one not likely, *a priori*, to be associated with air pollution). Next, we examined the impact of smoothers of time with varying spans (defined as the percent of observations included in each smoothing

“window”). Finally, we reexamined the models that showed associations between particulate matter and mortality after deleting days with 1%, 5% and 10% of the highest PM concentrations. This would minimize the impact of extreme observations and provide indirect evidence of the impact of PM10 on windy days.

The primary multivariate analyses were conducted in Stata (Stata, 1997) and SAS using PROC GENMOD (SAS, 1992).

## Results

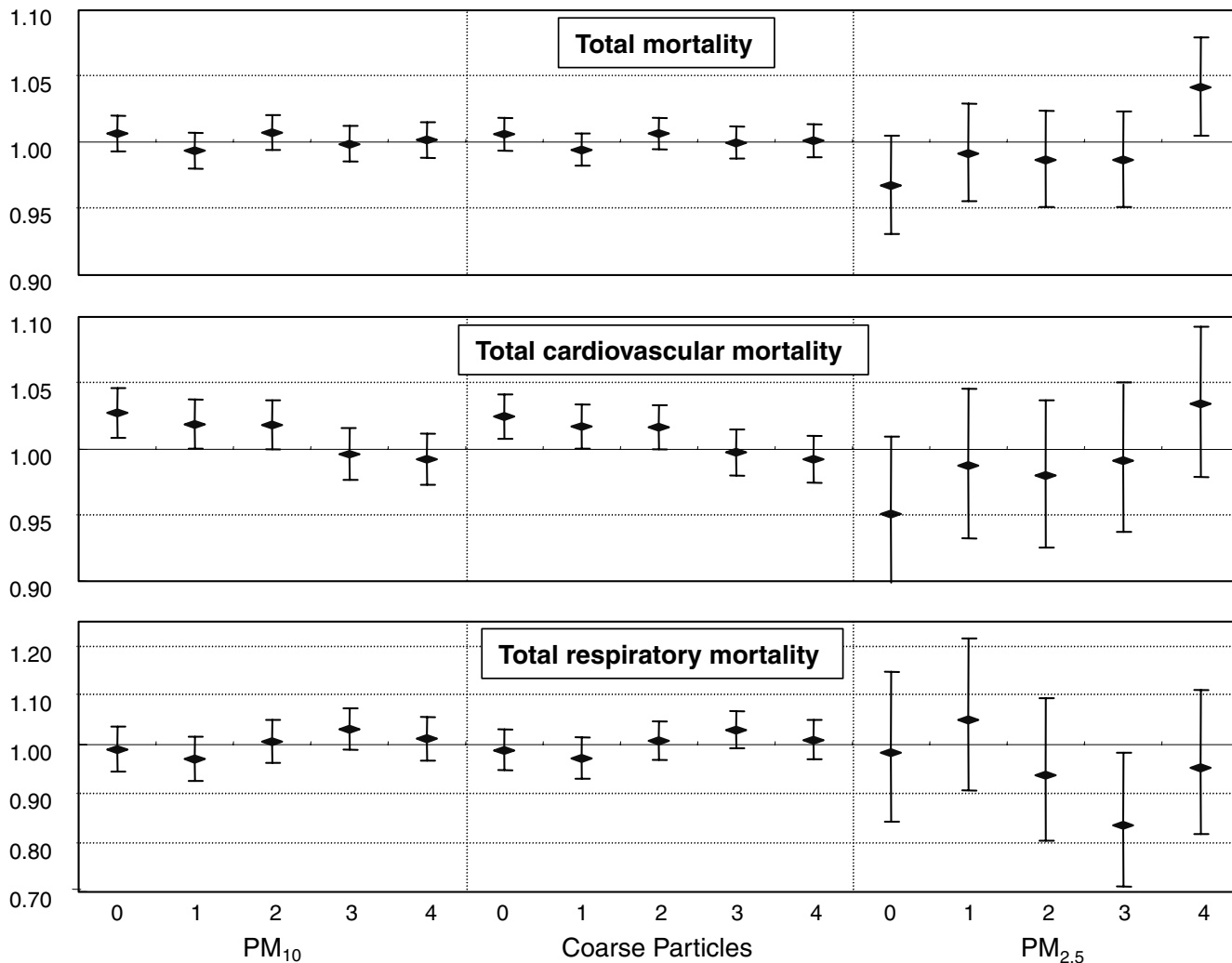
Table 1 indicates the means and ranges of the health endpoints, air pollution concentrations and meteorological variables. Mean daily mortality counts were 5.8 for all causes, 2.7 for cardiovascular and 0.52 for respiratory deaths. Total and cardiovascular deaths were slightly higher

**Table 2.** Correlations among pollutants and temperature in Coachella Valley, California, 1989–1998.

	PM10p	PM10i	CFp	CFi	PM2.5p	PM2.5i	NO <sub>2</sub>	Ozone	CO
PM10i	0.60								
CFp	0.94	0.69							
CFi	0.53	0.97	0.61						
PM2.5p	0.68	0.35	0.46	0.18					
PM2.5i	0.65	0.46	0.49	0.28	0.60				
NO <sub>2</sub>	-0.15	-0.14	-0.16	-0.21	-0.12	-0.02			
Ozone	0.42	0.24	0.33	0.20	0.41	0.43	-0.30		
CO	0.01	-0.04	0.02	-0.08	0.07	0.15	0.45	-0.18	
Temp	0.44	0.30	0.40	0.30	0.44	0.46	-0.32	0.75	-0.18

PM10i=PM10 at Indio; PM10p=PM10 at Palm Springs.; CFp=coarse particles at Palm Springs; CFi=coarse particles at Indio; PM2.5p=PM2.5 at Palm Springs; PM2.5i=PM2.5 at Indio; NO<sub>2</sub>=nitrogen dioxide at Palm Springs (24-h avg); Ozone=ozone at Indio (1-h max); CO=carbon monoxide at Palm Springs (8-h avg); Temp=maximum daily temperature at Palm Springs. All variables are unlagged.

### Relative risks for interquartile range for alternative lags of measures of particulate matter.



Note: Numbers at the bottom of the table indicate the lag, in days.

Figure 1. Relative risks for interquartile range for alternative lags of measures of particulate matter.

during the winter months and increased slightly over the 10-year period. Mean PM<sub>10</sub> (24-h average) concentrations were 47  $\mu\text{g}/\text{m}^3$  in Indio and 30  $\mu\text{g}/\text{m}^3$  in Palm Springs. The coarse fraction comprised 55% of PM<sub>10</sub> mass in Indio and 60% in Palm Springs, with concentrations of 26 and 18  $\mu\text{g}/\text{m}^3$ , respectively, while the corresponding concentrations of PM<sub>2.5</sub> were 17 and 13. Carbon monoxide and nitrogen dioxide levels were low, while ozone levels were moderate (average of 1-h maxima of 7 pphm in Palm Springs, with a high of 19) relative to current ambient standards. All pollutant levels exhibited some seasonal variation. PM<sub>10</sub> and CF were generally higher during the months of April through August, with the highest

concentrations occurring during July. PM<sub>2.5</sub> and ozone were highest during June through August, while carbon monoxide and nitrogen dioxide peaked during the winter months. The windiest months (based on average daily wind speed) were April through July.

Table 2 summarizes the correlations among different particulate matter metrics, gaseous pollutants, and mean temperature. For PM<sub>10</sub>, the correlation between the daily concentrations at Palm Springs and Indio during the study period was 0.60. PM<sub>10</sub> was more strongly correlated with CF ( $r=0.97$  at Indio and 0.94 at Palm Springs) than with PM<sub>2.5</sub> ( $r=0.46$  at Indio and 0.68 at Palm Springs). The correlation of PM<sub>10</sub> and ozone was 0.42 at Palm Springs.

**Table 3.** Regression results for total, cardiovascular and respiratory mortality for Coachella Valley, 1989–1998 (relative risks and 95% CI for interquartile change).

Pollutant description	IQR	Total mortality	Total cardiovascular mortality	Total respiratory mortality
PM10 (24-h avg, unlagged, $\mu\text{g}/\text{m}^3$ )	24.6	1.01 (0.99–1.02)	1.03 (1.01–1.05)	0.99 (0.94–1.04)
Coarse particles (predicted 24-h avg, unlagged, $\mu\text{g}/\text{m}^3$ )	19.6	1.01 (0.99–1.02)	1.02 (1.01–1.04)	0.99 (0.95–1.03)
PM2.5 (24-h avg, 4-day lag, $\mu\text{g}/\text{m}^3$ )	9.0	1.04 (1.00–1.08)	1.03 (0.98–1.09)	0.95 (0.82–1.11)
Ozone (1-h maximum, pphm)	4.0	0.99 (0.96–1.03)	0.96 (0.91–1.01)	1.03 (0.91–1.16)
Carbon monoxide (8-h avg, ppm)	0.5	1.03 (1.01–1.05)	1.00 (0.97–1.03)	1.03 (0.95–1.11)
Nitrogen dioxide (24-h avg, pphm)	1.1	1.03 (1.00–1.05)	1.02 (0.99–1.05)	1.01 (0.94–1.08)

Correlations between PM10 and both carbon monoxide and nitrogen dioxide were low. Finally, PM10 and maximum daily temperature were moderately correlated;  $r=0.30$  at Indio and  $0.44$  at Palm Springs. The smoothed plot of mortality and maximum temperature indicated an inverse, linear, but fairly flat relationship.

The best de-trended model developed to explain variations in mortality prior to the inclusion of pollutant variables included maximum temperature (unlagged), indicator variables for Monday and Saturday, and a loess smooth of time with 10 degrees of freedom. Subsequent results were insensitive to the degrees of freedom chosen for the smooth.

Figure 1 summarizes the RRs for total, cardiovascular, and respiratory mortality using alternative lags (0 to 4 days) for the measures of particulate matter. Table 3 summarizes the basic results. PM2.5 was associated with all-cause mortality (RR=1.04; 95% C.I., 1.00–1.08, 4-day lag evaluated at the interquartile range (IQR) of  $9 \mu\text{g}/\text{m}^3$ ). Among the gases, CO (unlagged, 8-h average; RR=1.03; 95% C.I., 1.01–1.05; IQR=0.52 ppm) and NO<sub>2</sub> (unlagged, 24-h average; RR=1.03; 95% C.I., 1.00–1.05; IQR=1.1 pphm) were also associated with total mortality. For the gaseous pollutants and PM2.5, these results were sensitive to the lag specification in that associations were not apparent for other lag structures.

For cardiovascular-specific mortality, associations were found for two of the particulate matter metrics. For PM10 and CF, lags of 0 to 2 days were all associated with cardiovascular mortality, with the unlagged value providing the strongest associations (PM10 RR=1.03; 95% C.I., 1.01–1.05; IQR=25  $\mu\text{g}/\text{m}^3$ ; CF RR=1.02; 95% C.I., 1.01–1.04; IQR=20  $\mu\text{g}/\text{m}^3$ ). No association with cardio-

vascular mortality was found for PM2.5 or any of the gaseous pollutants. When either PM10 or the coarse fraction was entered into the model for cardiovascular mortality along with any of the gaseous pollutants, the particulate measure retained a significant association with mortality. None of the pollutants was associated with respiratory mortality. Neither ozone metric (1- or 8-h average) was associated with any mortality outcome. None of the particle measures had a positive, significant association with mortality due to “other” causes (i.e., total minus cardiovascular and respiratory mortality).

Additional sensitivity analyses were conducted for particles and cardiovascular mortality. First, we examined the impact of using a four-day moving average of pollutant concentrations. Only for the relationships between PM10 and CF and cardiovascular mortality did the cumulative exposure make a difference; in these instances the RRs were slightly larger than those for any single-day lag (results not shown). Second, we tested the impact of smaller sample sizes. In contrast to the results using the 10 years of predicted values of CF, when the 2.5 years of CF data were run, no associations were found. We tested the power of the data by re-running the regressions using several randomly selected 2.5 years periods of the predicted value of CF. The resulting regression coefficients for CF were quite variable in their strength of association with mortality. In the third set of sensitivity analyses, summarized in Table 4, the highest 1%, 5%, and 10% of each of the measures of particulate matter were excluded from the analysis. Excluding the highest concentrations of PM10 and CF did not affect the associations of these pollutant metrics with cardiovascular mortality. PM2.5 was

**Table 4.** Regression results for cardiovascular mortality and selected data exclusions, Coachella Valley, 1989–1998 (RR and 95% CI for interquartile change).

Pollutant description	IQR	Full model (no exclusions)	Excluding highest 1%	Excluding highest 5%	Excluding highest 10%
PM10 (24-h avg, unlagged, $\mu\text{g}/\text{m}^3$ )	24.6	1.03 (1.01–1.05)	1.03 (1.00–1.05)	1.04 (1.01–1.08)	1.04 (1.00–1.08)
Coarse particles (predicted 24-h avg, unlagged, $\mu\text{g}/\text{m}^3$ )	19.6	1.02 (1.01–1.04)	1.03 (1.00–1.05)	1.04 (1.01–1.08)	1.04 (1.00–1.08)
PM2.5 (24-h avg, 4-day lag, $\mu\text{g}/\text{m}^3$ )	9.0	1.03 (0.98–1.09)	1.03 (0.98–1.09)	1.03 (0.97–1.09)	1.06 (0.99–1.13)

not associated with cardiovascular mortality and this result was not altered by subsequent data exclusions.

## Discussion

PM10 in the Coachella Valley is dominated by the coarse fraction. Based on 2.5 years of data collected in Palm Springs and Indio, the coarse fraction comprised 60% and 55% of PM10 mass, respectively. Daily correlations between PM10 and the coarse fraction were high in both areas:  $r=0.94$  in Palm Springs and  $0.97$  in Indio, while the correlations between PM10 and PM2.5 were  $0.68$  and  $0.46$ , respectively. Thus, daily variation in PM10 during the study period was driven primarily by changes in the coarse fraction, with fine particles exerting a lesser influence. This contrasts with conditions in metropolitan areas in the U.S., particularly those on the East Coast, where the fine fraction is more strongly correlated with PM10, and where the ratio of the coarse fraction to PM10 is approximately  $0.35$  (U.S. EPA, 1996). Analysis of 10 years of mortality data in the Coachella Valley indicates associations between cardiovascular-specific mortality and both PM10 and coarse particles. The magnitude of the estimated relative risk of cardiovascular mortality associated with PM10 is about  $1.1\%$  per  $10 \mu\text{g}/\text{m}^3$  PM10. This estimate is within the general range of cardiovascular mortality effects reported in previous studies ( $0.8\%$  to  $1.8\%$ ) and similar to our previous study in the Valley (U.S. EPA, 1996; Ostro et al., 1999). Unlike our prior study, however, there was no association between any particle metric and respiratory-related mortality. This may be due in part to the small proportion of total deaths in this investigation classified as respiratory. No association was found between PM2.5 and cardiovascular mortality. A less robust association was found between PM2.5 and all-cause mortality, where only a four-day lag in PM2.5 was statistically significant. The reduced sample size for PM2.5 ( $n=1020$  days) may have influenced the statistical power. Moreover, mean 24-h average PM2.5 concentrations in the Valley were low:  $13 \mu\text{g}/\text{m}^3$  in Palm Springs and  $17 \mu\text{g}/\text{m}^3$  in Indio.

Neither PM10 nor coarse particles were associated with the "control" measure of mortality (total minus cardiopulmonary), which supports the notion that the association of these particle measures with cardiovascular mortality was likely to have been causal. Among the gaseous pollutants, associations were found between both nitrogen dioxide (both 1- and 24-h average) and carbon monoxide (both 1- and 9-h average) and all-cause mortality. These associations were not affected by alternative treatments of time, season, or weather. However, only the unlagged specifications were statistically significant.

We examined the influence of higher concentrations of particulate matter. When days with the highest 1%, 5% and

10% of PM10 concentrations and coarse particles were excluded from the analysis, the coefficients increased slightly. Thus, it appears that the associations were not driven by the highest concentrations. Rather, there is evidence of a slight diminution of effect at the higher concentrations. Coarse particles, but not fine, are nearly linearly related to mean daily wind speed (although the highest concentrations of coarse particles do not occur on the windiest days). This suggests that there is a lesser impact on daily mortality on those days with the highest PM10 concentrations, which occur on relatively windy days. Thus, wind events *per se* are not likely to be strongly associated with mortality, and even suggest an attenuation of effect. This result is similar to findings reported by Pope et al. (1999) and Schwartz et al. (1999). Such results could be partly explained by greater time spent indoors, and consequent reduced exposure, on windy days.

There are relatively few epidemiological studies of the acute effects of coarse mode particles among the general population. Two studies have examined selected health outcomes in relation to dust storms in eastern Washington state (Hefflin et al., 1994; Schwartz et al., 1999). Schwartz et al. (1999) found no relation between total mortality on 17 days marked by dust storms in Spokane, compared with control dates during which there were no dust storms. This finding is not inconsistent with our findings, as noted above. The study by Hefflin *et al.* examined relationships between days on which PM10 levels exceeded  $150 \mu\text{g}/\text{m}^3$  and hospital emergency room (ER) visits. Those investigators found PM-associated increases in ER visits for bronchitis and sinusitis (both less than  $1\%/10 \mu\text{g}/\text{m}^3$ ), and none for asthma. Pope et al. (1999) investigated the relative impacts of PM10 on mortality in three cities in Utah with different PM10 source profiles, including geologic particles from windblown dust. They found that in Salt Lake City (but not Ogden or Provo/Orem), some of the PM10-mortality associations were strengthened when episode days dominated by high levels of windblown dust were dropped from the model, suggesting that the combustion particles more commonly associated with air stagnation were more strongly related to daily mortality than crustal particles. These results are not entirely consistent with ours; however, we also found that our results were not driven by the PM levels found on the windiest days.

Several other studies have also recently found effects of coarse particles on mortality and morbidity. Castillejos et al. (2000) reported that coarse particles in Mexico City were more strongly associated than fine particles with total, cardiovascular, and respiratory mortality in Mexico City. Van den Eeden *et al.* have found that cardiovascular hospital admissions in Los Angeles are also associated more strongly with coarse than with fine particles (S. van den Eeden, personal communication, 1999). Examining outpatient visits for upper and lower respiratory conditions in relation

to ambient PM<sub>10</sub> in Anchorage, AL, Gordian et al. (1996) reported a 3% to 6% increase in outpatient visits for asthma and a 1% to 3% increase for sinusitis visits associated with a 10- $\mu\text{g}/\text{m}^3$  increase in ambient PM<sub>10</sub>. In Anchorage, much of the PM<sub>10</sub> is in the coarse fraction, originating from volcanic ash and soil.

The mechanisms that underlie the associations between exposure to PM and mortality in elderly people are unknown. Data from several toxicological and epidemiological investigations provide suggestive evidence that a variety of mechanisms could be operative, which might well vary by particle source, composition, and size distribution. Preexisting pulmonary inflammation could facilitate PM-induced release of proinflammatory mediators, resulting in additional pulmonary inflammation, bronchoconstriction, hypoxia and cardiac effects, including ventricular fibrillation and death (Godleski et al., 1996). Seaton et al. (1995) proposed that exposure to ultrafine particles could provoke alveolar inflammation, causing exacerbations of existing lung disease and increased blood coagulability, leading in turn to cardiovascular deaths through direct effects on either the lungs or the heart. More recently, Seaton et al. (1999) found suggestive evidence of red blood cell sequestration related to PM<sub>10</sub> concentrations, possibly attributable to increased RBC adhesive properties. Individuals with compromised coronary circulations could therefore be at increased risk of PM-associated occlusive episodes. Peters et al. (1997) reported a particle (TSP)-associated shift in the upper tail of the distribution of plasma viscosity, suggesting that inflammation in the lung might influence blood rheology and extrapulmonary vascular events. Using concentrated ambient particles, Godleski et al. (1996) produced airway inflammation and pulmonary vascular congestion in rats with chronic bronchitis. Alterations in cardiac electrophysiological function, including PR- and ST-segment changes, were observed in dogs exposed to concentrated ambient air particles (Godleski et al., 1997).

As with the epidemiological literature, there are few toxicological data examining acute effects of coarse particles other than silica, which produces a prolonged inflammatory response in experimental animals at high exposure concentrations (Warheit et al., 1991; IARC, 1997). Prior special monitoring studies by the South Coast Air Quality Management District (SCAQMD) indicate that approximately 10% of the annual average PM<sub>10</sub> mass consists of silicon, a substantial portion of which is in the form of silica (SCAQMD, 1990; Mel Zeldin, South Coast Air Quality Management District, personal communication, 1998). In an experiment examining the relative acute effects of inhalation of nitrates, sulfates, and coarse particles (produced by resuspending road dust) in the lungs of rats, Kleinman et al. (1995) found that all three exposures produced some effects consistent with lung injury, though those investigators inferred that the two

fine-particle exposures were generally more potent in inducing such effects than the resuspended road dust. Both sulfate and road-dust exposures caused a significant suppression of alveolar macrophage function, while increased epithelial permeability was observed in rats exposed to road dust or to nitrates, but not sulfates (Kleinman et al., 1995). To the extent that coarse particles are capable of provoking or exacerbating a pulmonary inflammatory response, it is possible that they may, like fine particles, elicit effects on the cardiovascular system remote from the site of particle deposition.

The possibility of such effects was suggested in a recent report examining the effects on human monocytes exposed *in vitro* to extracts of coarse and fine particles collected in North Carolina (Monn and Becker, 1999). Those authors reported that water-soluble extracts derived from coarse particles induced significantly greater cytotoxicity and approximately 20-fold greater quantities of the proinflammatory cytokines IL-6 and IL-8 than did extracts from fine particles collected concurrently. Monn and Becker attributed their findings to the presence of both endotoxin and various transition metals in the coarse fraction.

In summary, in this investigation we found consistent associations between two measures of ambient particles (PM<sub>10</sub> and the coarse fraction) and daily cardiovascular mortality. This relationship persisted throughout numerous sensitivity analyses and was not observed for noncardio-pulmonary mortality. Although this investigation was carried out in an area in which PM<sub>10</sub> is strongly correlated with the coarse fraction (consisting mainly of crustal particles), the magnitudes of the associations are similar to those observed in numerous urban areas in which variability in particle concentration is due principally to changes in combustion-related fine particles. Recent findings by other groups are consistent with ours and suggest that, at least in some locations, the coarse fraction may play as important a role in PM-related toxicity as fine particles.

### Disclaimer

This manuscript has been reviewed and approved for publication. However, the opinions and conclusions expressed are those of the authors and do not necessarily represent those of the California Office of Environmental Health Hazard Assessment, the South Coast Air Quality Management District, or the California Public Health Institute.

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