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Wildfire air pollution and daily mortality in a large urban area

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Abstract

Unusual air pollution episodes, such as when smoke from wildfires covers a large urban area, can be used to attempt to detect associations between short-term increases in particulate matter (PM) concentrations and subsequent mortality without relying on the sophisticated statistical models that are typically required in the absence of such episodes.

The objective of this study was to explore whether acute increases in PM concentrations from wildfire smoke cause acute increases in daily mortality. The temporal patterns of daily nonaccidental deaths and daily cardiorespiratory deaths for June of 2002 in the Denver metropolitan area were examined and compared to those in two nearby counties in Colorado that were not affected by the wildfire smoke and to daily deaths in Denver in June of 2001. Abrupt increases in PM concentrations in Denver occurred on 2 days in June of 2002 as a result of wildfire smoke drifting over the Denver area. Small peaks in mortality corresponded to both of the PM peaks, but the first mortality peak also corresponded to a peak of mortality in the control counties, and cardiorespiratory deaths began to increase on the day before the second peak. Further, there was no detectable increase in cardiorespiratory deaths in the hours immediately following the PM peaks.

Although the findings from this study do not rule out the possibility of small increases in mortality due to abrupt and dramatic increases in PM concentrations from wildfire smoke, in a population of over 2 million people no perceptible increases in daily mortality could be attributed to such events.

Keywords: Air pollution; Mortality; Particulate matter; Forest fires; Cardiovascular disease

1. Introduction

Short-term increases in particulate matter (PM) air pollution concentrations have often been associated with acute increases in daily mortality in urban settings (Samet et al., 2000; Burnett et al., 2000; Katsouyanni et al., 1997). The associations are typically based on time series studies in which correlations between time series of daily PM concentrations and daily death counts are examined, while attempting to control for meteorological and other time-varying factors that potentially confound the associations. Uncertainties about the adequacy of the temporal control and the adequacy of the specification of meteorological factors have raised concerns that estimates of PM effect from these time series studies may still be biased due to residual confounding (Health Effects Institute, 2003). One alternative approach to assessing acute effects of PM that does not necessarily rely on time series modeling and the inherent uncertainties in the use of these models is to exploit unusual air pollution episodes that potentially avoid confounding by either temporal trends or meteorology.

In this study, we have attempted to make use of two such unusual air pollution episodes that occurred when abrupt shifts in wind direction caused smoke from a large wildfire in Colorado to drift over the large urban area of metropolitan Denver. Although smoke from wildfires often affects residential communities, it is uncommon for large urban areas to be affected. The abrupt increase in PM

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concentrations associated with the smoke and the large size of the population exposed combined to suggest that the hypothesis that acute exposure to fine, combustion-derived PM causes increased mortality could be tested without reliance on time series analyses.

2. Methods

2.1. Mortality data

Counts of daily total deaths for 2001 and 2002 for the Denver metropolitan region (Adams, Arapahoe, Denver, Douglas, and Jefferson Counties) and for two control counties also in the Front Range of Colorado having sizeable populations (El Paso and Larimer Counties (Fig. 1)) were obtained from the Colorado Health Information Data set compiled by the Health Statistics Section of the Colorado Department of Public Health and Environment. Mortality data from Weld County, another Front Range county with a sizeable population, were not used since visibility data (see Air pollution and meteorological data) indicated that Weld County experienced smoky days in June when Denver had smoky days. ICD-10 codes were used to identify and exclude accidental deaths (ICD-10 letter prefixes V, W, X, Y, and Z) and to stratify deaths into cardiorespiratory causes (ICD-10 letter prefixes "I" and "J") and noncardiorespiratory causes. Data on gender and age at death were also available.

2.2. Air pollution and meteorological data

Air pollution and meteorological data for June of 2001 and 2002 were obtained from the Air Pollution Control Division (APCD) of the Colorado Department of Public Health and the Environment. The APCD operates and maintains a network of monitoring sites for Colorado. For the Denver metropolitan region, only the daily air pollution data from the central monitoring site located in downtown Denver were used. The only other APCD hourly PM monitoring site in the five-county Denver metropolitan region in 2002 was located in an industrial area in northwestern Denver; concentration data from this site roughly paralleled those from the central site and are therefore not displayed. For the two control counties, the daily PM data from the single APCD monitoring site in each county, some of which were available only every 3 days, were used. Hourly airport visibility data from the National Climatic Data Center were also collected for the two control counties.

3. Results

Smoke from the large Hayman fire southwest of Denver drifted over the Denver area on June 9th and June 18th in 2002 and blanketed the entire region (Fig. 1). This resulted in abrupt and dramatic, but short-lived, concentration increases in both inhalable (PM smaller than 10 μm in diameter [PM_{10}]) and fine PM (PM smaller than 2.5 μm in diameter [$PM_{2.5}$]) on those days (Fig. 2A). Peak 1-h concentrations of PM_{10} and $PM_{2.5}$ were 372 and 200 $\mu g/m^3$, respectively, on June 9th and 316 and 200 $\mu g/m^3$, respectively, on June 18th (Fig. 3). The dramatic increase in PM concentrations lasted from 4 to 5 h on each day. Twenty-four-hour average concentrations of PM_{10} and $PM_{2.5}$ were 91 and 44 $\mu g/m^3$, respectively, on June 9th and 88 and 48 $\mu g/m^3$, respectively, on June 18th. The fraction of PM_{10} made up of $PM_{2.5}$ on the smoky days was generally higher than that on the nonsmoky days in June (Fig. 2A), indicating that the proportion of fine particles in the inhalable fraction of PM was higher.

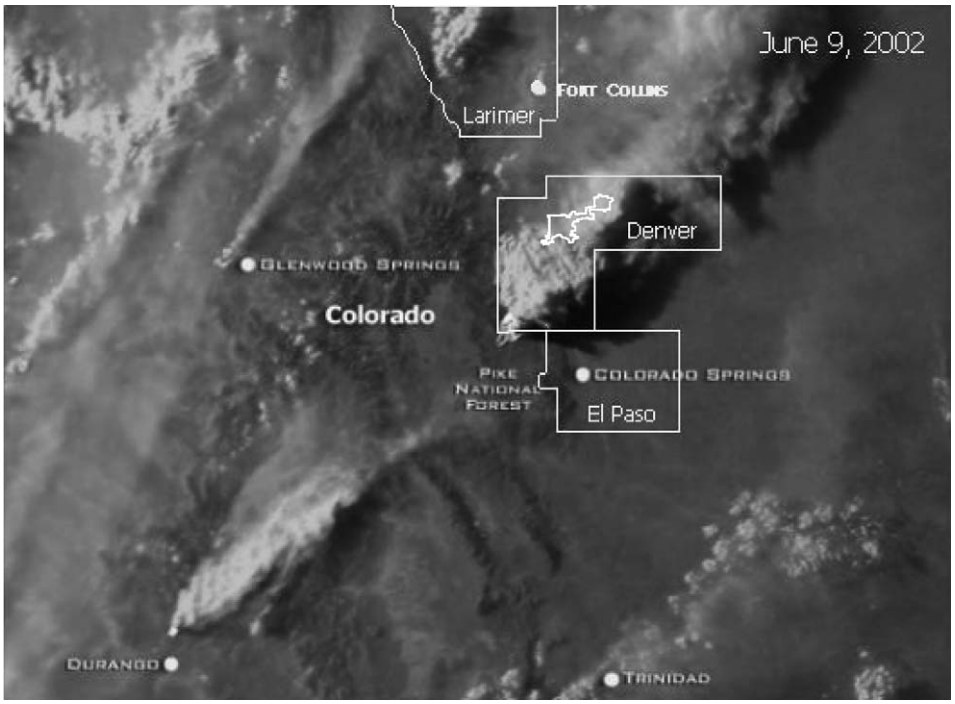

Fig. 1. National Oceanic and Atmospheric Administration satellite photo (http://www.osei.noaa.gov/Events/Fires/US_West/2002/FSMHSus-CO161_N5.jpg) shows the smoke from the Hayman fire southwest of Denver blanketing the Denver area on June 9, 2002. The locations of the five-county Denver metropolitan region ("Denver"), with Denver County outlined within, and the two comparison counties ("Larimer" and "El Paso") are also shown. The Hayman fire is at the lower left corner of the "Denver" region.

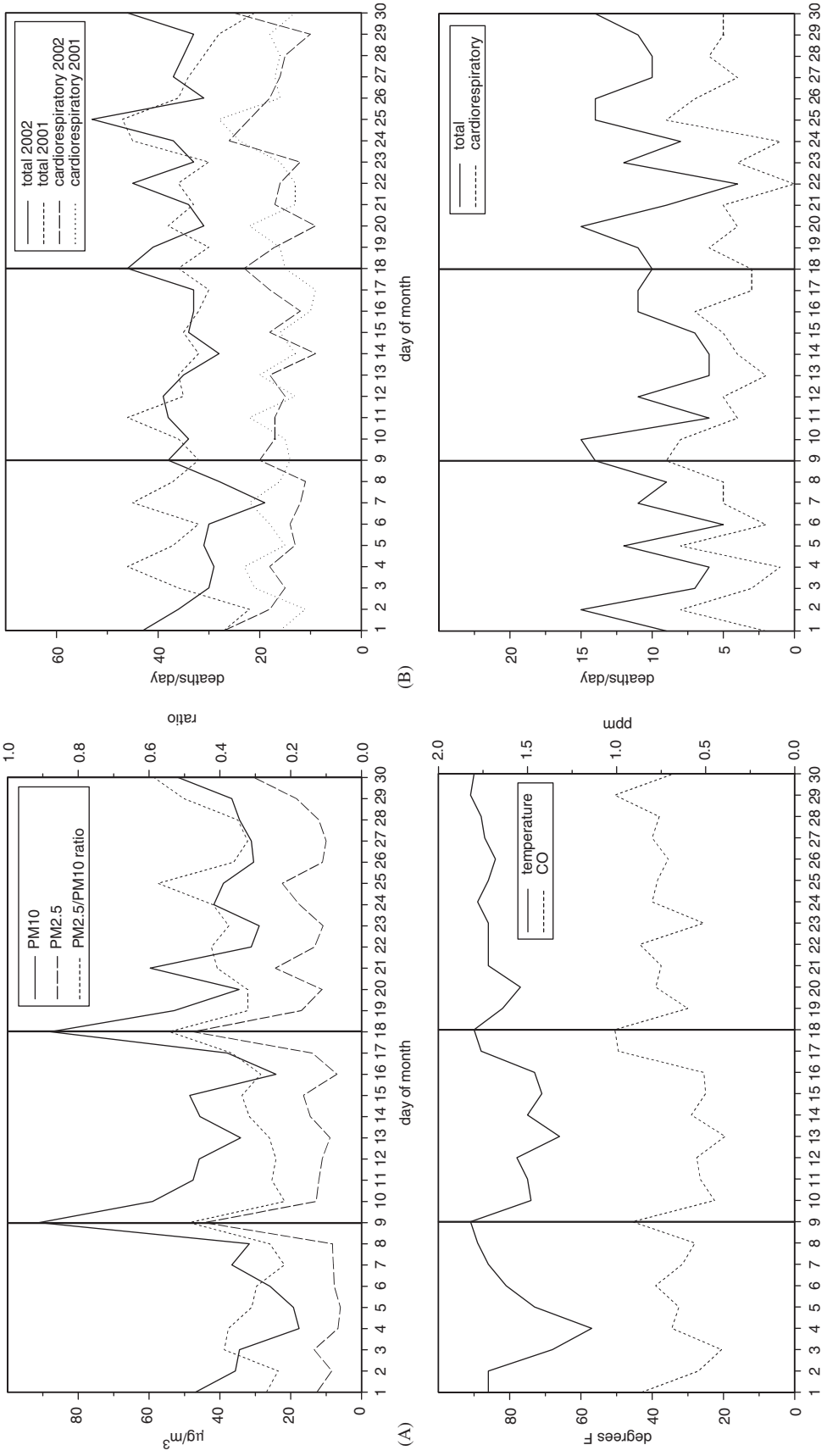

Fig. 2. June 2002 air pollution, meteorology, and daily deaths in Denver. (A) Mean 24-h (from 12 AM to 12 AM) PM_{10} and $PM_{2.5}$ concentrations ($\mu g/m^3$) showing the marked increase in concentrations in Denver and the increase in the $PM_{2.5}/PM_{10}$ ratio on the 2 wildfire smoke days, June 9th and June 18th, 2002 (vertical lines). (B) Daily total nonaccidental and cardiorespiratory mortality in June 2002 and, by comparison, 2001 showing peaks in mortality on the two smoky days in 2002 (vertical lines) and no significant difference in raw death counts in June of 2001 and 2002. (C) Daily maximum hourly temperature (degrees F) and 24-h average carbon monoxide (CO) concentrations (ppm) in June 2002 exhibited peaks on the smoky days (vertical lines). (D) Pattern of daily deaths in June 2002 for the two control counties that did not experience wildfire smoke on either the 9th or the 18th showed a peak on June 9th but not on June 18th.

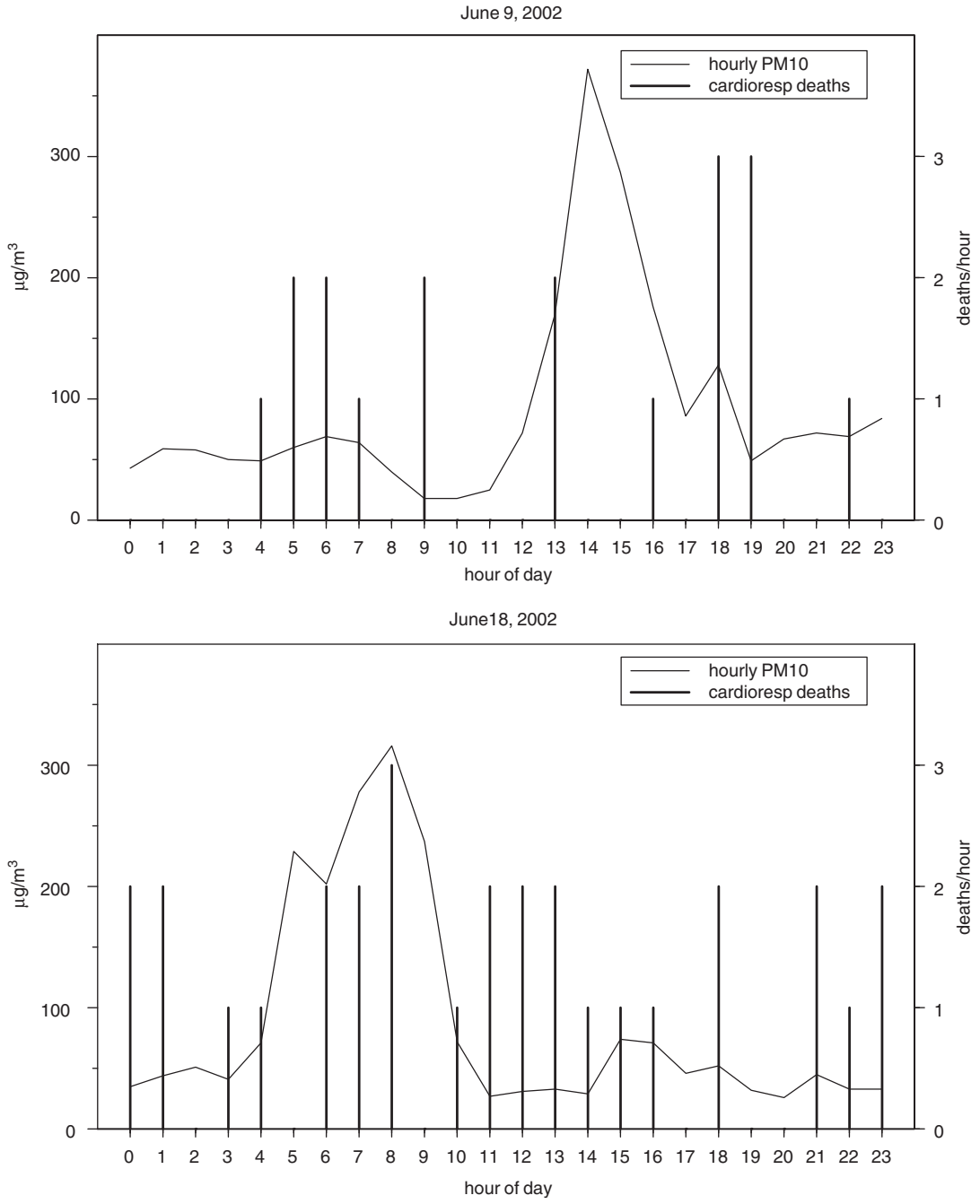

Fig. 3. Hourly PM_{10} concentrations ($\mu g/m^3$) on the 2 wildfire smoke days in June 2002 show the dramatic increases in concentrations that occurred during the day on those 2 days. Shown also are the hourly number of cardiorespiratory deaths (cardioresp deaths) for those days accumulated over the hour of each PM_{10} concentration measurement.

Total nonaccidental deaths in the Denver metropolitan region averaged 35.3 (SD 6.7) deaths per day for the month of June in 2002; deaths in which the underlying cause of death was coded as cardiorespiratory averaged 16.6 (SD 4.7) deaths per day in June. There were small peaks in daily total nonaccidental deaths and daily cardio-respiratory deaths on both June 9th and June 18th, but these were not unusual for the month (Fig. 2B). For cardiorespiratory deaths, the increase in deaths around the second smoke event began 1 day before the increase in PM concentrations. No increases in daily deaths in the few days following these high pollution days were apparent. The median age of those who died on or shortly after the 2 smoky days was not higher than that on other days in June (data not shown). Focusing more specifically on the smoky days, no abrupt increase in deaths was apparent in the hours immediately following the smoky hours (Fig. 3).

Maximum daily temperature peaks were also present in June of 2002, and two of these corresponded to the PM-associated mortality peaks (Fig. 2C). Concentrations of

carbon monoxide also peaked on the 2 wildfire smoke days (Fig. 2C). Peak 1-h carbon monoxide concentrations on the 2 days were 2.1 and 3.0 ppm, respectively, and these corresponded to the hours when PM concentrations peaked on those days.

Daily deaths from the two control counties combined also showed daily mortality peaks, with one occurring on June 9th, the first of the smoky days in Denver (Fig. 2D). No wildfire smoke affected these counties on that day; neither the PM concentration data that were available for that day nor the visibility data indicated that PM concentrations were increased in these counties on that day. There was no peak in mortality in the control counties that corresponded to the second smoky day in June in Denver.

Daily deaths were comparable in June of 2001, a month when wildfire smoke did not drift over Denver, as in June of 2002 (Fig. 2B). In particular, the mean daily total and cardiorespiratory death counts from the 9th to the end of June 2001 (16.0 and 7.6 per million population, respectively) were similar to those from the 9th to the end of June 2002 (16.9 and 7.7 per million, respectively). Meteorology was similar in June of 2001 and 2002, with mean maximum daily temperature being 80.1 (SD 9.1) and 81.3 (SD 8.6)°F in June of 2001 and 2002, respectively.

4. Discussion

Based on a newspaper report, at least one death, in a woman with asthma who lived close to the fire, was suspected to be directly attributed to smoke from the Hayman fire (Sherry, 2002). However, no acute increase in daily deaths could be attributed to the abrupt and dramatic increases in PM concentrations from the Hayman wildfire smoke in a large urban region of over 2 million people. Some of the several peaks in daily deaths in June of 2002 seemed to correspond in time to the 2 smoky days. The first peak (June 9), however, was also seen in the control counties that did not experience an increase in PM concentrations on those days, implying that some other phenomenon, perhaps the peaks in temperature that also corresponded to those days, was responsible. Furthermore, the peak in PM concentrations on the first smoky day occurred at 2 P.M., so that if the mortality peak were in fact due to the smoke, the increase in deaths from the smoke would have had to occur in the 10 h remaining in that day. No such acute effect was apparent. Peters et al. (Peters et al., 2001) observed an increase in myocardial infarction within hours of increases in PM concentrations, but the link to acute cardiac events has not been confirmed in other studies (Sullivan et al., 2002). Peters et al. (2004) also recently reported a threefold increase in risk of myocardial infarction within 1 h of using some form of transportation. The lack of a clear acute effect of wildfire smoke on acute cardiorespiratory deaths in the hours following a dramatic increase in PM concentrations raises the possibilities that the effect of transportation in the Peters report was unrelated to traffic-associated pollution or that traffic-associated pollution is more toxic than wildfire smoke. It is instructive to recall that in the dramatic air pollution episode in London, England in December of 1952, an episode that started in the early morning, only a small increase in daily deaths was apparent on the first day (Logan, 1953), indicating that an increase in deaths on the same day as that of the dramatic increase in PM concentration would not necessarily be expected.

The second smoke-associated mortality peak (June 18) was not seen in the control counties. However, on this occasion the subset of total deaths due to cardiorespiratory causes, the subset of deaths deemed to be most clearly associated with increases in PM concentrations, began to increase 1 day before the smoke event, although they increased further on the smoky day. Therefore, on neither of the 2 smoky days was there good evidence that the mortality peaks were due to the increase in PM concentrations on those days.

Estimates of increases in daily total nonaccidental deaths typically range from 1.1% (Dominici et al., 2003) to 8.3% (Fairley, 2003) for each 50-$\mu g/m^3$ increase in 24-h average PM_{10} concentration; for daily cardiorespiratory deaths, estimated increases range from 1.6% (Dominici et al., 2003) to 9.5% (Mar et al., 2003). For the approximately 40 total nonaccidental deaths per day that occur in the Denver region, an excess of from 0.4 to 3.3 deaths per day due to the 50-$\mu g/m^3$ increase in 24-h average PM_{10} concentration observed in Denver in June 2002 on each of 2 days would have been expected. For the approximately 20 daily cardiorespiratory deaths in the Denver region, an excess of from 0.3 to 1.8 deaths per day due to the 50-$\mu g/m^3$ increase in 24-h average PM_{10} concentration would have been expected. Given the day-to-day variability in deaths over a month in a large urban area, such predicted excesses in deaths would not have been perceptible using the crude comparisons employed here to identify an effect of wildfire smoke. However, it is unlikely that the 24-h average increase in PM_{10} in Denver adequately reflects the impact of the peak increases in hourly PM, for which the maximum increase was approximately 350 $\mu g/m^3$ on June 9th. At the other extreme, if such an increase in PM concentration over several hours were assumed to be equivalent to a 24-h increase of that magnitude, the estimated excess deaths would range from approximately 3–23 excess total deaths per day and from 2 to 13 excess cardiorespiratory deaths per day. Increases at the upper end of these ranges should have been detectable by the crude comparisons made here.

While there is currently no consensus with regard to which part of the above ranges is the more valid, estimates from most multicity studies congregate at approximately 3% increased daily total nonaccidental deaths for each 50-$\mu g/m^3$ increase in 24-h average PM_{10} concentration or 21% for a 350-$\mu g/m^3$ increase (Burnett and Goldberg, 2003; Schwartz, 2003; Katsouyanni et al., 2003). For the PM concentration increases experienced in Denver in June of

2002, the latter estimate corresponds to 11 expected excess daily deaths. Such increases in daily deaths were seen in June of 2002 in Denver in the absence of pollution concentration increases, indicating that the ability to detect effects on mortality from events that cause dramatic PM concentration increases may require similar data from urban areas substantially larger than Denver.

A reasonable argument has been made that the impact of short-term increases in PM concentration would not be expected to be limited to 1 day but would instead extend over several days (Schwartz, 2000). We therefore might have expected that the increases in daily deaths would have extended over more than 1 day. Such a pattern in the PM-related mortality peaks was not seen. Further, based on comparison of daily deaths in June of 2001 and 2002, there was no indication that daily deaths were higher than usual in the days following the smoke events.

In addition to the possibility that even in an urban area as large as Denver there is limited power to detect effects of dramatic air pollution events, at least four other arguments can be put forward to attempt to explain why an increase in deaths could not be detected. First, it is possible that abrupt, but short-lived, increases in PM concentrations from combustion sources do not in fact acutely cause mortality. The estimates of increased mortality associated with short-term increases in PM concentrations are based on observational studies in which attempts are made, by statistical modeling, to control for all of the time-varying factors other than PM concentrations, such as meteorology and other air pollutants, that could confound the temporal association between PM and mortality. Because the estimates of PM effect are very small, control for these confounding factors must be very good to prevent residual confounding. There is currently no agreement with regard to the best approach to use in controlling for these other factors and even more importantly, how aggressive should be the attempt to control for them in the statistical models (Health Effects Institute, 2003). The quasiexperimental nature of this study allows many, if not all, of these time-varying factors to be controlled by design, rather than through statistical modeling. The uncertainties inherent in statistical modeling are thus avoided. If in fact "natural" experiments such as this do not confirm the effects estimated from the models, concern over residual confounding in the time series studies is heightened.

Second, it has been argued that the deleterious effect of PM is a function of chemical composition and that PM from combustion sources is more harmful than PM from crustal or other noncombustion sources (Pope et al., 1999; Schwartz et al., 1999). PM in smoke from wildfires is obviously a product of combustion. However, it is possible that only certain types of combustion produce toxic particles. For example, fossil fuel combustion emissions typically contain metal-containing PM that in toxicologic studies has been shown to be more toxic than PM that does not contain metals (Campen et al., 2002). To the extent that wildfire PM does not contain these particles, it is possible that PM from wildfires is less toxic.

Third, it is possible that the temporal profile of PM concentrations on the smoky days was distinct enough from daily temporal PM profiles in time series studies that similar effects would not be expected. The duration of each smoky event in Denver lasted for only part of 1 day. This temporal pattern is quite distinct from the typical day-to-day variability in relatively low PM concentrations seen in urban areas on which time series estimates of PM effect are based. While there are advantages to examining effects of a discrete, high concentration event in countering effects of temporally confounding factors that plague time series studies, the relevance of such events to the effects of the more typical urban air pollution temporal profiles might be questioned.

Fourth, no effect of smoke events on mortality might be expected if susceptible individuals took effective measures to avoid exposure. Clearly the presence of the wildfire smoke was obvious to the residents of Denver. Once this became obvious, it is likely that people went indoors and that air conditioning use increased. However, being indoors does not necessarily lessen exposure to smaller, and potentially more toxic, particles; particles smaller than 1 μm in diameter gain ready access to indoor air (Janssen and Zuidhof, 1998). On the other hand, in at least one study the effects of PM were lessened in households with air conditioning (Janssen et al., 2002), suggesting that air conditioner use might ameliorate the effects of increases in ambient PM concentrations.

Since large urban areas only rarely experience substantial air pollution from wildfire smoke, data such as those presented here are unusual. Singapore experienced dramatic increases in PM concentrations from wildfire smoke from late August to early November in 1997 that originated from wildfires largely in Indonesia. While there was an increase in medical clinic and emergency room attendances for respiratory complaints in association with increases in PM concentrations in Singapore, no increases in hospitalizations or in mortality were reportedly observed (Emmanuel, 2000), a finding in agreement with that from Denver. In contrast, effects of Indonesian wildfire smoke on increased mortality in Malaysia have been reported (Sastry, 2002). The recent wildfires in southern California in the fall of 2003 and the wildfires in Quebec, Canada in the summer of 2002, smoke from which affected large urban areas in the northeastern United States (DeBell et al., 2004), might also provide opportunities to investigate effects of increased PM concentrations from wildfire smoke on mortality and other outcomes.

In conclusion, although the findings from this study do not rule out the possibility of small increases in mortality due to abrupt and dramatic, but brief, increases in PM concentrations from wildfire smoke, in a population of over 2 million people no perceptible increases in daily mortality could be attributed to such events. This indicates, at the least, that if there is an effect of such increases in PM concentration from wildfire smoke on mortality, the effect

is a relatively small one. Since mortality is the most adverse of the adverse health effects attributed to air pollution, it is possible that the wildfire smoke caused increases in less adverse health effects in Denver, as suggested by findings on the effects of Indonesian wildfire smoke on Singapore (Emmanuel, 2000). In this regard, further investigation of the effects of wildfire smoke on less adverse outcomes than mortality would be valuable.

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