Episodes of High Coarse Particle Concentrations Are Not Associated with Increased Mortality

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Fine particle concentration (i.e., particles <2.5 µm in aerodynamic diameter; $PM_{2.5}$), but not coarse particle concentration, was associated with increased mortality in six U.S. cities. Others criticized this result, arguing that it could result from differences in measurement error between the two size ranges. Fine particles are primarily from combustion of fossil fuel, whereras coarse particles (i.e., particles between 2.5 and 10 µm in aerodynamic diameter) are all crustal material, i.e., dust. One way to determine if coarse particles are a risk for mortality is to identify episodes of high concentrations of coarse, but not fine, particles. Spokane, Washington, is located in an arid area and is subject to occasional dust storms after crops have been harvested. Between 1989 and 1995, we identified 17 dust storms in Spokane. The 24-hr mean PM_{10} concentration during those storms was 263 µg/m³. Using control dates that were the same day of the year in other years (but with no dust storm on that day) and that had a mean PM_{10} concentration of 42 µg/m³, we compared the rate of nonaccidental deaths on the episode versus nonepisode days. There was little evidence of any risk [relative risk (RR) = 1.00; 95% confidence interval (CI), 0.81–1.22] on the episode days. Defining episode deaths as those occurring on the same or following day as the dust storm produced similar results (RR = 1.01; CI, 0.87–1.17). Sensitivity analyses, which tested more extensive seasonal control, produced smaller estimates. We conclude that coarse particles from windblown dust are not associated with mortality risk. *Key words:* air pollution, dust storms, mortality, particulates. *Environ Health Perspect* 107:339–342 (1999). [Online 23 March 1999]
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During evolutionary time the particles inhaled by lungs were primarily the products of mechanical processes; they are generally >2.5 µm in aerodynamic diameter and are commonly referred to as coarse particles. Substantial human exposure to combustion particles only began with the domestication of fire, and these particles differ from coarse particles in size, source, and physicochemical properties. They are <2.5 µm in diameter (and usually <1 µm), referred to as fine particles, and are more likely to deposit in the alveolar region of the lung. For example, the EPA estimated (1) [based on the International Commission of Radiological Protection model (2)] that the fraction of the total aerosol mass deposited in the alveolar region that derives from particles <2.5 µm in diameter ranges from 98% in locations with aerosols similar to Philadelphia to 80% for aerosols similar to Phoenix.

Fine particles also contain higher concentrations of sulfates and nitrates, organic compounds, and more bioavailable transition metals than coarse particles (3,4). Until 1997, the EPA regulated concentrations of particles in the air <10 µm in aerodynamic diameter (PM_{10}), without regard to source, size, or chemical composition. Many of the U.S. counties exceeding the EPA standard do so because of episodes of wind-blown dust. Public officials in these counties complained that they were forced to take expensive measures because the EPA inappropriately included coarse particles in the standard. In 1997, the EPA added regulation specifically targeted to combustion particles, concluding that they were probably more toxic. This conclusion was based in part on a study by Schwartz et al. (5), who reported that fine particles, but not coarse particles, were associated with daily deaths is six U.S. cities. Lipfert and Wyzga (6) criticized this study, arguing that greater measurement error in the coarse particle measurements could have weakened the coarse particle association.

The thesis that combustion particles are associated with increased deaths is supported by studies of episodes of high concentrations of combustion aerosols. Studies from London in 1952 (7) demonstrated that very high concentrations of combustion-associated air pollution were associated with substantial increases in daily deaths. These high air pollution concentrations occurred in a period of low wind speed and thus low concentrations of windblown dust. An episode in western Germany in 1985 (8) reported a substantial increase in deaths associated with lower, but still high, levels of particulate matter. While the concentrations in these episodes were high, they provide some assurance that the associations

seen at more common levels are causal, particularly because they appear to lie on the same dose–response curve (9).

Similarly, assessment of the health effects of coarse particles could be aided by examining episodes of high concentrations of coarse particles. Indeed, for coarse particles, episode studies have a major advantage. Episodes of high concentrations of combustion particles are characterized by low wind speed and a low-lying inversion layer, which leads to increases in other combustion-related pollutants as well as particles. Hence, the studies pinpoint combustion pollution as a risk factor for mortality, but it is more difficult to determine which species of the combustion mix is responsible.

In contrast, episodes of high coarse mass particles are generally associated with dust storms, that is, conditions of high wind speeds that tend to diminish the concentrations of fine particles and all other combustion-related pollutants. This allows us to unambiguously attribute any pollution effect. By focusing on such episodes, the issue of measurement error is moot because the contrast is between days with unambiguously high levels of coarse particles and control days with much lower levels. We applied this approach to data from Spokane, Washington, a county in a semiarid region of the west, which is occasionally subject to dust storms.

Data and Methods

Data

Air pollution data. Air pollution data were obtained from the Spokane Air Quality Control Board (SCAPA). We identified dust storms using lists compiled by Haller et al. (10), SCAPA, and National Climatic Data Center Weather Observations. We confirmed those days by examining data on PM_{10} concentrations and carbon monoxide (CO) concentrations in Spokane. Dust

storm days would be expected to have very high PM_{10} levels and high wind speeds, but low levels of CO, which is a combustion product. In all, we identified 18 dust storm episodes between 1989 and 1996. These are listed in Table 1, along with environmental data on those days. All PM_{10} data were taken from Crown Zellerbach, which was the only location with daily reports throughout the period.

Mortality data. Data on deaths in Spokane County were obtained from death certificate records filed with the State Department of Health for the years 1989–1995. These were summarized into daily counts of deaths from nonexternal causes [*International Classification of Diseases*, Revision 9 (ICD-9) <800]. Because no data were available for 1996, the analysis was limited to 17 dust storms.

Methods

In the primary analysis, deaths on the day of a dust storm were contrasted with deaths on control days. A sensitivity analysis defined deaths following exposure to be those occurring on the day of the dust storm or the following day. Including the day after the dust storm allowed us to accommodate a lag between exposure and response. In the London episode of 1952

(6), for example, mortality rose on the day pollution rose, but seemed to peak somewhat after the peak of exposure.

Control days were chosen to be the same day of the year in other years, when dust storms did not occur on that day. For the sensitivity analysis, control days were defined as the same day of the year as the dust storm day or the following day, in other years, provided dust storms did not occur on those days. This provides for seasonal control by matching. In total, there were 95 control days chosen in the primary analysis and 171 in the sensitivity analysis.

The rate ratio for being a dust storm day was estimated in a Poisson regression, controlling for temperature, dew point temperature (a measure of humidity), and day of the week dummy variables. Sensitivity analyses were also done to assess the impact of using a linear time trend variable or of using dummy variables for year of study.

Mortality varies seasonally, and the matching strategy assumes that the choice of the same day of the year controls for this variation. However, the seasonal pattern in mortality can change from year to year. While on average we would expect this to cancel out, in a finite data set, it may not. We tested the sensitivity of our analyses to this by filtering the data to remove seasonality

and by repeating the analysis contrasting exposure and control days using the adjusted daily deaths after control for season. To control for season, we fit a generalized additive Poisson regression to the daily death counts from 1989 to 1995. The predictor variables used were day of the week dummies and a smoothed function of time, which captures the seasonal pattern and allows for different seasonal patterns in different years (11). The smoothing parameter was chosen to minimize the partial autocorrelation function of the data. By removing the seasonal pattern, we reduced the fluctuations in the data to "white noise," which has no serial correlation.

Results

Table 2 shows the average values of PM_{10}, CO (maximum hourly), temperature, and dew point temperature on exposure and control days. The temperature and dew point temperatures were closely matched, in addition to being controlled in the regression analysis. Average PM_{10} levels on exposure days were 221 $\mu g/m^3$ higher than those on control days. In contrast, CO levels were lower during the exposure periods, which is consistent with the fact that high wind conditions tend to reduce combustion-related pollutants. We operated a particulate monitor in Spokane during 1996, with a size cutoff of 1 μm. Essentially all the particles captured by this monitor were from combustion sources. There was one dust storm in 1996, on 30 August; the mean PM_{10} concentration during that storm was 187 $\mu g/m^3$, but the PM_1 concentration was only 9.5 $\mu g/m^3$. The mean PM_1 concentration for all of August was 10.4 $\mu g/m^3$, and the mean PM_{10} concentration was 47 $\mu g/m^3$. This confirms that dust storms are associated with elevations in crustal particles but not in combustion particles.

The mean daily death count was slightly lower on the exposure days than on control days whether we used only the dust storm day or included the day following the storm in the definition. Hence, univariately, no risk is seen for exposure to high concentrations of crustal particles.

Using previous studies, we estimated that an incremental PM_{10} exposure of 221 $\mu g/m^3$ would be associated with approximately a 20% increase in daily mortality, if coarse particles were equally toxic with the combustion particles that dominated the variation in PM_{10} in the earlier studies. Our results are shown in Table 3. We found no evidence of increased mortality on these high PM_{10} days [relative risk (RR) = 1.00; 95% confidence interval (CI), 0.81–1.22].

The risk of mortality on dust storm days after controlling for linear time trend (RR for dust storm = 0.99; CI, 0.81–1.22),

Table 1. Dust storm days in Spokane, Washington, 1990–1996

Date	Temperature (°F)	Dew point	Wind gusts (mph)	PM_{10} ($\mu g/m^3$)	Max 1-hr CO (ppm)
8 September 1990	72	47	21	217	6.37
12 September 1990	64	45	25	112	3.40
4 October 1990	59	46	46	342	3.15
9 November 1990	50	43	48	268	2.45
23 November 1990	47	38	56	309	2.50
13 September 1991	59	36	46	147	4.60
16 October 1991	52	29	62	NA	2.10
21 October 1991	50	33	52	351	2.20
4 September 1992	58	37	41	321	3.43
12 September 1992	56	30	37	803	1.80
13 September 1992	49	24	32	163	1.65
25 September 1992	55	33	30	110	2.95
26 September 1992	56	37	45	252	4.30
8 October 1992	53	28	30	175	3.85
11 September 1993	56	35	37	300	1.88
3 November 1993	45	28	39	207	5.33
24 July 1994	86	50	32	129	2.10
30 August 1996	72	48	34	187	2.85

Abbreviations: PM_{10}, particulate matter ≤10 μm in aerodynamic diameter; CO, carbon monoxide; NA, not available.

Table 2. Mean environmental levels and daily deaths on exposure and control days in Spokane, Washington, 1990–1996

	Deaths on day of dust storms		Deaths on day of and day after dust storms	
Variable	Exposure days	Control days	Exposure days	Control days
PM_{10} ($\mu g/m^3$)	263	42	192	43
Temperature (°F)	57	54	55	53
Dew point (°F)	36	38	35	38
Max CO (ppm)	3.2	5.3	3.8	5.4
Deaths	7.52	7.96	7.66	7.83

Abbreviations: PM_{10}, particulate matter ≤10 μm in aerodynamic diameter; CO, carbon monoxide.

Table 3. Relative risk (RR) and 95% confidence intervals (CI) for dust storm days by model specification

Model	RR	CI
Basic model	1.00	0.81–1.22
Time trend	0.99	0.81–1.22
Yearly dummies	0.99	0.80–1.23
Day after storm	1.01	0.87–1.17
Day after storm plus time trend	1.01	0.87–1.17
Day after storm plus yearly dummies	0.98	0.84–1.14
Filtering out season	0.94	0.78–1.14

and yearly dummy variables (RR for dust storm = 0.99; CI, 0.80–1.23) both showed no increased mortality. When we extended the analysis by considering deaths on the day of the dust storm or the day following the storm, similar results were found. The exposure contrast was 149 $\mu g/m^3$, but for all cause mortality, there was no association between exposure and daily deaths (RR for dust storm = 1.01; CI, 0.87–1.17). Controlling for linear time trend or year of study again did not change these results.

When we first filtered out seasonality using a generalized additive model and then contrasted adjusted deaths on the exposed and control days, we still failed to find any evidence of an association (RR for dust storms = 0.94; CI, 0.78–1.14).

Discussion

We found no evidence that mortality was elevated on dust storm days in Spokane, Washington. The number of dust storms and the average number of deaths per day influenced the confidence intervals of the findings. However, the fact that in all the different models only one had a regression coefficient greater than zero more strongly suggests that crustal particles have little toxicity. We have no measurements of $PM_{2.5}$ during this period, so the parallel study, directly confirming that combustion particles are toxic, cannot be done in Spokane. However, two recent studies have associated PM_{10} with hospital admissions for respiratory (12) and cardiovascular (13) disease during this time period. Those studies were restricted to days when PM_{10} concentrations were below 150 $\mu g/m^3$. Hence, they excluded the dust storms analyzed here and focused on days when much of the PM_{10} in Spokane was from combustion sources. This provides indirect evidence that noncrustal particles in Spokane are toxic.

These results are consistent with the previous literature on air pollution and mortality. The great air pollution episodes of the midcentury [London in 1952 (6), Donora, Pennsylvania, in 1948 (14), and the Meuse Valley in Belgium in 1930 (15)] were all episodes of combustion-related fine particles that occurred in stagnant air conditions which resulted in low dust levels. Most studies of the adverse effects of particles have been conducted in urban areas where fine combustion particles are the major source of daily variability in particle concentration. Thus, evidence primarily reflects fine particles and tells us little about coarse mass.

Following the Mount St. Helens eruption, which produced very high concentrations (10,000 $\mu g/m^3$) of coarse particles, few adverse health effects were seen. Buist et al. (16) followed 101 children that attended a summer camp which was significantly impacted by the eruption. They found little evidence for an effect of exposure either over time or on the evening-to-morning differences following a day of activity in air that exceeded the then-current particulate standard of 260 $\mu g/m^3$. Similarly, a study of a dust storm in southeastern Washington State found little impact from an episode in which particle concentrations exceeded 1,000 $\mu g/m^3$ (17). These results are consistent with our findings.

Most animal studies have targeted combustion particles and have recently reported important evidence for toxicity. For example, Godleski et al. (18,19) exposed rats with chemical-induced bronchitis or chronic lung inflammation to concentrated fine particles from the Boston, Massachusetts, air. While little effect was seen in healthy rats, the rats with lung disease had substantial mortality. In a second study, electrocardiogram patterns in dogs exposed to concentrated fine particles showed arrhythmic changes, which were enhanced by coronary occlusion (20,21). Zelikoff et al. (22) exposed rats infected with streptococcal pneumonia to concentrated fine particles from New York City air; they reported a doubling of the region of the lung with pneumonia in exposed animals as compared to controls. However, these studies tell us little about the potential toxicity of coarse particles.

Osornio-Vargas et al. (23) assessed the toxicity to lung cells of particles sampled from different areas of Mexico City. The particles from the region where combustion particles dominated were much more toxic than the particles from the region where wind blown dust was also an important contributor to PM_{10}.

Dreher and colleagues (24,25) examined particle toxicity in vivo by size range of particle. They showed that the pulmonary toxicity of urban particles varied with size, with the greatest toxicity in particles <1.7 μm and the least toxicity in those >3.5 μm. Several factors may contribute to this differential toxicity.

Most of the particles that deposit in the alveolar region are fine particles (1). Clearance rates for particles in the alveolar region are much slower than in the conducting airways, which may be one reason for the greater toxic effect of fine particles.

Other factors may also contribute to the lower toxicity of coarse particles. Costa and Dreher (25) reported that urban particles have substantial amounts of soluble transition metals and produce pulmonary toxicity. In contrast, Mount St. Helens particles had less soluble transition metals and showed less toxicity when instilled at the same dose (26). Washington, DC, particles >3.5 μm in diameter also had less soluble transition metals.

Transition metals play a role in the particle toxicity. Li et al. (27) and Gilmour et al. (28) instilled 50–125 μg of particles removed from PM_{10} filters in Edinburgh, Scotland, into rat lungs. They reported increased recruitment of neutrophils into the lung, and that cultured bronchoaveolar lavage cells produced excess quantities of tumor necrosis factor α and macrophage inflammatory protein 2, which are proinflammatory cytokines. Pretreatment with a metal-chelating agent substantially reduced the inflammatory response to the urban particles, documenting the substantial role of metals in the inflammatory process. Dreher et al. (29) reported similar findings.

The role of chemical composition in particle toxicity is still poorly understood, but there is evidence that metals may not be the only characteristic of importance. For example, Peters et al. (30) reported a stronger association between ultrafine particle concentrations and respiratory health than for other particle measures. Brunekreef et al. (31) reported that truck traffic exposure was a better predictor of children's respiratory health and suggested that diesel particles with more organic particulate (and also ultrafine concentration) may be more toxic. Here too, the candidates characteristics are more typical of fine particles than coarse crustal particles.

In summary, the results of our study are consistent with other human and animal studies of coarse particles and indicate that toxicity of coarse particles is substantially less than that of fine particles. This finding, in a study where measurement error is not an issue, supports the conclusion that control of airborne particles should focus on combustion particles if the goal is to reduce the impact on human health. It also indicates that dust storms are not a significant threat to respiratory and cardiovascular health and that regulatory efforts should not be focused on dust storms.
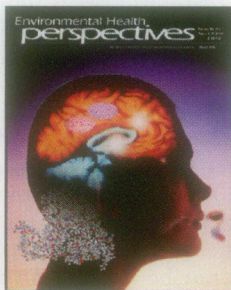
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