CASE STUDY

The Effect of Urban Air Particulates on Human Mortality

The question of whether particulate air pollution actually does cut human lifespan by increasing mortality rates has been intensively researched in the past few decades, particularly in the United States. Epidemiological studies have shown consistent and significant associations between daily and long-term mortality rates and airborne particulate concentrations in the air, especially the fine component ($PM_{2.5}$). These correlations are consistently much stronger than are those with the concentrations of any gaseous pollutant.

In such studies, the rates of death—either total rates or rates from diseases such as lung cancer or cardiovascular disease—are plotted against the average concentration of particulates in order to determine whether they are related to each other. In epidemiology, the existence of an approximately linear correlation between two quantities x and y does not *prove* that x causes y (or vice versa)—for example, that breathing suspended particles causes mortality. It may be that the group studied was small, and it was by chance that the variables correlated. Several repetitions of the study, using larger groups and correcting for variables other than x that could conceivably affect y, are often required to convince scientists that a *causal* relationship exists. A possible complication in such studies is that it is actually another variable z that causes y to vary, but z usually varies in the same way as does x in many environments, so it appears that x is the driving variable. For example, the SO_2 concentration and the fine particulate level are closely correlated in cities in which coal is burned as a fuel, so health effects that appear to correlate with one may in fact be caused by the other. The uncertainty as to the real driver can be overcome by including in the study locations in which the two quantities are known *not* to be closely related. Still another test of whether a correlation of y with x is likely to be causal or not is to consider whether such a relationship "makes sense" with respect to the biology involved.

The most influential study of the effect of air pollution, both acute and chronic, on human health was carried out for six medium-sized American cities using data over three periods, extending from the late 1970s to the late 1990s. The cities were Portage, Wisconsin; Topeka, Kansas; Watertown, Massachusetts; Harriman, Tennessee; St. Louis, Missouri; and Steubenville, Ohio. Initially, there were 8,111 adults in the study that began in the mid-1970s. The main conclusion from the Six-Cities studies is that high levels of fine particulate pollution, $PM_{2.5}$, causes the mortality rate to rise in a city on the same day as the pollution occurs and for some time thereafter. It was also established that the annual mortality rate in the six cities increases in proportion to the yearly average $PM_{2.5}$ level.

The correlation between the *daily* death rate and $PM_{2.5}$ concentration that resulted from the Harvard Six-Cities study is shown in Figure 1. The shaded area in the figure represents the uncertainty in the various data points. The best straight line through the data goes close to the origin, indicating that there is no threshold: Any amount of fine particles in the air increased the mortality rates. The plot implies that typical U.S. cities in this period with high $PM_{2.5}$ ($\sim$20 $\mu g/m^3$) had about a 1.4% higher daily death rate

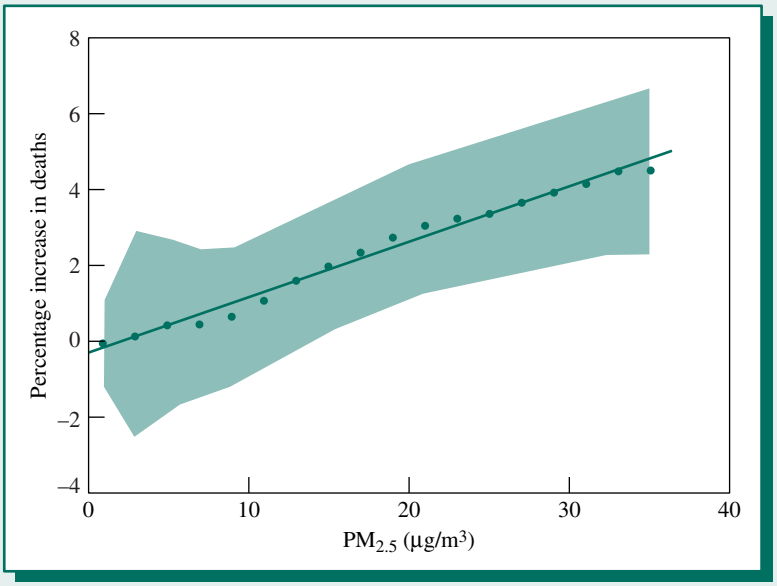

FIGURE 1 Overall estimated does–response relation between total $PM_{2.5}$ and daily deaths in six U.S. cities. The estimate is obtained by combining the estimated smoothed curves in each of the cities, after controlling for weather, season, and day of the week. The shaded area indicates the pointwise 95% confidence intervals at each point. The line shown is a least-squares regression line through the estimated points. [Source: J. Schwartz et al., "The Concentration–Response Relation Between $PM_{2.5}$ and Daily Deaths," *Environmental Health Perspectives* 110(2002): 1025.]

than typical low $PM_{2.5}$ (~10 $\mu g/m^3$) cities due to short-term air pollution. A reduction of 10 $\mu g/m^3$ across the board would result in about 36,000 fewer early deaths per year in the United States, about the same number that die in automobile accidents.

In the Six-Cities study, the greatest increase in mortality arose from particles that originated from vehicle emissions, followed by those—mainly sulfates—resulting from coal combustion. Fine particles originating from dust and soil had no effect on mortality. However, at the time the original results were gathered, leaded gasoline was still in use, so the composition and/or effects of particles from automobiles may have changed since then. There are hints in the data and in other studies that ultrafine particulates are especially dangerous to health. Indeed, some scientists have warned that the drive to decrease $PM_{2.5}$ levels will be counterproductive if by doing so the number of ultrafine particles is greatly increased—for example, by converting from diesel to natural gas vehicles. Even gasoline engines produce more ultrafine particulates than do diesels.

A Canadian study found that short-term increases in fine-particle concentrations affected mainly people having acute lower respiratory diseases, chronic coronary artery diseases (especially the elderly), and congestive heart failure, but not a number of other conditions, including chronic upper respiratory diseases and acute coronary artery disease. A recent study

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covering 21 U.S. cities established a linear correlation, with zero threshold, of heart attack frequency among elderly people with the day's PM_{10} level, with the effect doubling for adults with pre-existing lung disease. Several other studies in North America have established that daily mortality rates, including that of very young children who succumb to sudden infant death syndrome, correlate with PM_{10} values. Particulates and gaseous pollutants have also been linked to an increase in fatal strokes.

The annual death rates in the Six-Cities study was also found to correlate linearly with the average annual particulate pollution levels. The overall death rate increased about 15%, with rates for mortality due to lung cancer and cardiovascular diseases each increasing by about 27%, for each increase of 10 $\mu g/m^3$ in annual $PM_{2.5}$. The pollution rates have fallen significantly in these cities over the quarter-century since the study began, and this has been reflected in a drop of mortality rates from $PM_{2.5}$, especially from cardiovascular disease and especially in the most-polluted cities. Thus some of the chronic effects of air pollution may be partially reversible over extended periods. On the other hand, decades-long exposure to pollutants was not found to be necessary to develop most of the increases in mortality risk, although the relative contributions to risk from elevated acute and chronic exposures are not clear.

A number of other studies involving a large number of American cities and towns have been reported concerning the effects of chronic particulate air pollution on overall mortality rates. Particulate levels correlated with long-term mortality rates from lung cancer and cardiopulmonary causes, but sensibly not from death from all other causes combined, in the most detailed of the analyses. In the most recent many-city study, a 4% increase in a city's overall mortality rate, due to 6% and 8% increases in death rates from cardiopulmonary causes (respiratory and cardiovascular) and lung cancer, respectively, were observed for every 10 $\mu g/m^3$ increase in the $PM_{2.5}$ index. The health risk from breathing particulate-polluted air is comparable to that of a nonsmoker living with a cigarette smoker or of being moderately overweight.

A number of analyses have also been performed to determine what subsets of the population, if any, are most susceptible to particulates in the air; the results from these studies are shown in Table 1. The elderly, infants, and those with certain pre-existing diseases are found to be the most susceptible to acute exposures to high pollution levels, though many of the deaths in these cases may be advanced only by a few days by the temporarily high pollution level. There is no evidence that deleterious effects of long-term pollution are restricted to these groups. Lifetime exposure to high particulate levels appears to reduce average life expectancy by 1–3 years; by contrast, smoking decreases it by about 9 years. Although people who already are sick or frail, and consequently whose date of death may be advanced only slightly by high levels of particulate air pollution, probably constitute many of its fatal victims, there is evidence from the Six-Cities study that other groups are affected as well, and that the effects of acute pollution on a specific day are not all immediate.

TABLE 1 Summary of Who's Susceptible to Adverse Health Effects from PM Exposure and Overall Health Relevance

Health Effects	Who's Susceptible?	Overall Health Relevance
Acute exposure		
Mortality	Elderly, infants, persons with chronic cardiopulmonary disease, influenza, or asthma.	Obviously relevant. How much life shortening is involved and how much is due to short-term mortality displacement (harvesting) is uncertain.
Hospitalization/other health care visits	Elderly, infants, persons with chronic cardiopulmonary disease, pneumonia, influenza, or asthma.	Reflects substantive health impacts in terms of illness, discomfort, treatment costs, work or school time lost, etc.
Increased respiratory symptoms	Most consistently observed in people with asthma and children.	Mostly transient effects with minimal overall health consequences, although for a few there may be short-term absence from work or school due to illness.
Decreased lung function	Observed in both children and adults.	For most, effects seem to be small and transient. For a few, lung function losses may be clinically relevant.
Plasma viscosity, heart rate, heart rate variability, pulmonary inflammation	Observed in both healthy and unhealthy adults. No studies of children.	Effects seem to be small and transient. Overall health relevance is unclear, but may be part of pathophysiologic pathway linking PM with cardiopulmonary mortality.
Chronic exposure		
Increased mortality rates, reduced survival times, chronic cardiopulmonary disease, reduced lung function	Observed in broad-based cohorts or samples of adults and children (including infants). All chronically exposed potentially are affected.	Long-term, repeated exposure appears to increase the risk of cardiopulmonary disease and mortality. May result in lower lung function. Population average loss of life expectancy in highly polluted cities may be as much as a few years.

Source: C.A. Pope III, "Epidemiology of Fine Particle Air Pollution and Human Health: Biologic Mechanisms and Who's at Risk?" *Environmental Health Perspectives* 108, Supplement 4 (2000): 713.

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Discussion Questions

1. Plots of death rates against SO_2 and NO_2 concentrations in outdoor air in the Six Cities also produced graphs that are approximately linear, but the points on average lie farther from the best straight line through them. In other words, statistically these correlations were not as quantitative as that with $PM_{2.5}$. Is there reason to believe that the concentration of these gases would probably correlate with those of fine particulate matter? If so, is it is possible that they are acting as the variable z discussed above?

2. Some scientists do not believe that the case has yet been proven concerning the causal link between particulate air pollution and human mortality. They point out that most people spend most of their time indoors, and that consequently their personal exposure to particulates is not tightly linked to outdoor pollution levels. In addition, no biological mechanism has yet been firmly established to account for the effect of the particles upon health. Do you agree with their position? Do you believe that particulate air pollution levels should be tightly controlled even in the absence of definitive proof of their association with increased mortality?