

Human Health: Effects of Ambient Air Particulate Matter

Morton Lippmann – New York University School of Medicine

ABSTRACT

The clearly identifiable human health effects of ambient air pollution follow their inhalation into the respiratory tract. In eastern North America, most of the acidic air pollution is associated with sulfuric acid aerosol, which is formed in the atmosphere as ultrafine particles ($d < 0.1 \mu\text{m}$) through the oxidation of sulfur dioxide emitted during fossil fuel combustion, and which is gradually agglomerated into fine particles ($0.2 < d < 1.0 \mu\text{m}$) and also neutralized to ammonium sulfate by reaction with ammonia gas, a product of anaerobic decay at ground level. Short-term peak exposures to ambient fine particles can cause asthma exacerbations and affect the lungs ability to clear itself of inhaled particles. Long-term chronic exposures are closely associated with premature mortality, increased rates of emergency department and hospital admissions, respiratory symptoms, and lost-time from work and school.

BACKGROUND

The quantitative associations between health and air pollution can be traced back to England in the 1950s and, until the mid-1980s, we had most

of our health effects defined by the experience in London and other parts of the United Kingdom.

Beauty is in the eye of the beholder. Claude Monet spent three years in London – 1901 to 1904 – and produced over 100 oil paintings of the London scene because he was fascinated by the interplay between the diffused light and the coal smoke particles. There were periodic pollution episodes in the late nineteenth and early twentieth centuries where many people died during the heavy London fogs. However, prior to the December 1952 episode, the public health data were quite limited.

The December 1952 episode couldn't be ignored because, in retrospect, it was determined that 4,000 people had died. During this fog episode people couldn't see more than a few feet in front of their faces. Furthermore, the effects were not limited to humans; prize cattle died at the Smithfield cattle show, and their lung edema was attributed to the acidic nature of the fog aerosol. There was a royal commission report (Ministry of Health, 1954) that led to the U.K. Clean Air Act, which banned soft coal burning, leading to an initial replacement with coke, and later with natural gas.

Figure 1 illustrates data from the last London

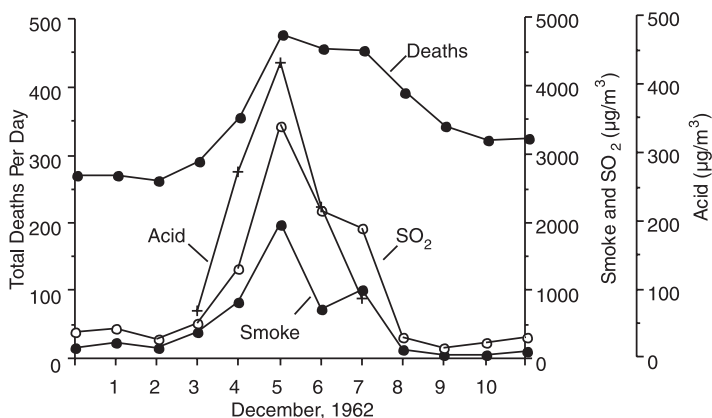


Figure 1. December 1962 London pollution episode.

SESSION III. Acid Rain Impacts: State of the Science

fog episode (December 1962) where one didn't need complex statistical analyses to see that there was an association between pollution, as it was being measured, and mortality. In the 1962 episode, the rise of the pollution was also clearly associated with a rise in the daily death rate, but the number of excess deaths was only about 700. The typical London fog pollution episodes lasted a few days. As in the previous mortality episode 10 years earlier, the death rate didn't come down as promptly as the pollutant concentrations. So, there was acute mortality with a temporal lag of a few days to a week or more, i.e., some people who were fatally affected didn't succumb immediately.

The 1962 fog episode provided the only data from that era where they had the daily measurements of sulfuric acid, as well as their classic indices of electrical conductivity of a scrubber solution (expressed as SO_2), and of particles, expressed as Black Smoke (blackness on a filter paper). The analyses suggested, as did the analyses of the earlier episode, that what was causing mortality was the strong acidity associated with the reaction, in the fog droplets, of SO_2 being oxidized and converted to sulfuric acid.

Beginning in the mid-1980s, in the United States, we began to see published reports of some epidemiological studies that have provided the basis for our newer thinking about the influence of ambient air particles on health. They provided a basis for the revised ambient air standards for the particulate matter (PM) in ambient air that I will review later in this presentation.

CURRENT UNDERSTANDING OF AMBIENT AIR PARTICULATE MATTER

Let us consider the physical aspects of the situation that we are dealing with. The sulfur dioxide (SO_2) gas that is emitted from a power plant stack moves downwind, and its concentration diminishes gradually with time and distance. One factor accounting for reduced concentration is the ground uptake of SO_2 on vegetation and other surfaces. Another factor is aerosol formation. What does that mean? It means that there is oxidation of the SO_2 to form SO_3 , a highly hygroscopic vapor.

Since there is always plenty of water vapor in the air, very fine (ultrafine) droplets of sulfuric acid form in huge numbers, over a million per milliliter. They rapidly coagulate, producing fine particles having diameters between about 0.1 and 1 μm that have a long atmospheric lifetime.

Thus, there is a persistent airborne source of SO_2 that creates fresh acidic aerosol over a period of days and hundreds or even thousands of kilometers downwind, with concentrations that are relatively uniform on a regional basis. This is illustrated in Figure 2, which depicts the results of some ambient air sampling conducted by NYU for another purpose. Hydrogen and sulfate ions were measured on a daily basis in the aerosol in both Buffalo and Rochester, NY over a six-week period. These cities are downwind of numerous power plants in the midwestern United States.

The figure shows that, over this 60-mile separation, there was a close correspondence in both sulfate ion and hydrogen ion concentrations, and that the measurements made in these two cities were as close as you would get with co-located samplers, i.e., running two samplers side-by-side. Thus, we can conclude that these fine particles are pervasive and widespread, at least over relatively flat land, and we don't need a lot of monitors in a metropolitan region to determine the sulfate ion concentration.

In fact, the fine particles, as represented by sulfate ion, are about 90 percent as high in most indoor environments as outdoors, because fine particles penetrate indoors. Thus, we are not just exposed to outdoor generated fine particles outdoors, but indoors as well; this helps us understand why epidemiology using outdoor measurements are relevant to exposures that may occur in indoor environments.

Now, let's consider the nature of airborne particles. Figure 3 is a depiction of the particle mass distribution. When we measure particles distribution according to their diameters in micrometers (aka microns), we find that most of the individual particles are very small ($< 0.1 \mu\text{m}$). However, these very numerous ultrafine particles contribute very little to the mass of the airborne particulate matter (U.S. EPA, 1996).

Most fine particles (diameters between 0.1 and 2.5 μm) were formed in the atmosphere from

Morton Lippmann - Human Health: Effects of Ambient Air Particulate Matter

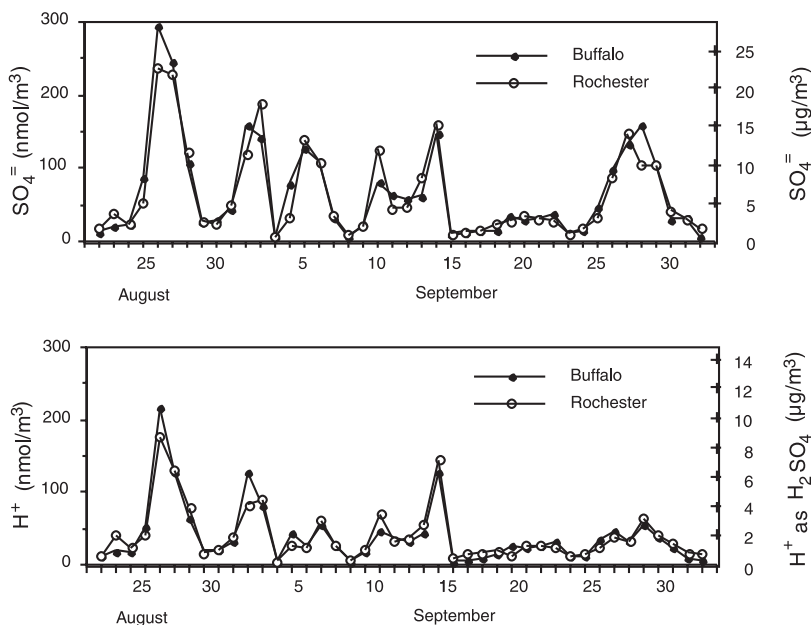


Figure 2. Intercomparison of Rochester, NY and Buffalo, NY sulfate and daily acid aerosol concentrations (August 22 – October 2, 1990).

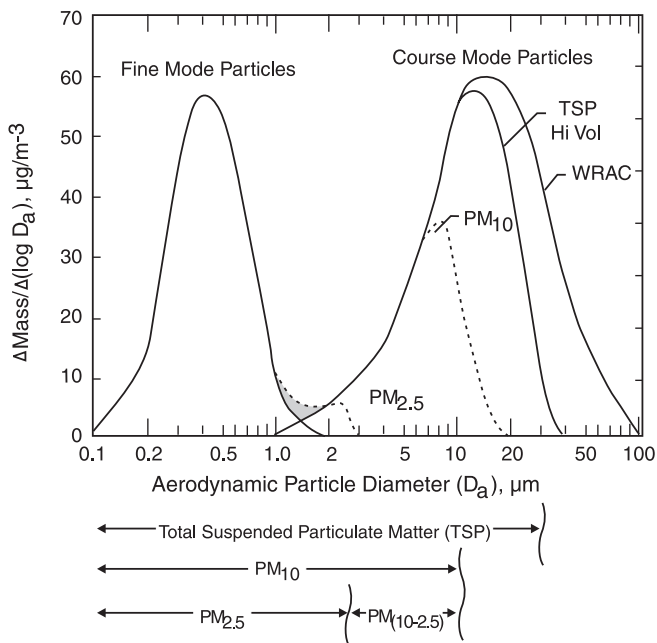


Figure 3. Representative bimodal mass distribution as a function of aerodynamic particle diameter for Phoenix, Arizona, showing effect of size-selective sampling inlet on mass collected for (a) wide-ranging aerosol classifier (WRAC), (b) standard total suspended particulate (TSP) high-volume sampler, (c) sampler following EPA's (PM₁₀) criteria for thoracic dust, and (d) sampler following EPA's criteria for fine particulate matter (PM_{2.5}). (Source: U.S. EPA, 1996).

SESSION III. Acid Rain Impacts: State of the Science

gaseous precursors as ultrafine particles. Following the coagulation of the ultrafines, which takes place within about a half hour of their formation, the resulting aerosol has a mass median diameter of about 0.3 to 0.5 μm , with only a small fraction of fine particles having diameters above one micron. However, when we consider the total surface area of the particles in the ambient air, most of it is in the fine particle mode.

When we look at Figure 3, which shows aerosol distribution in terms of mass, which varies

with the cube of diameter, we see a bimodal distribution. The coarse mode particles are soil-like and are produced by mechanical processes. The fine mode particles, which dominate in terms of surface area, are known as the accumulation mode because particles in this size range are too small to deposit by sedimentation, and too large to continue to grow by coagulation.

The chemistry of the two mass modes is radically different. As indicated in Table 1, the coarse particles are essentially soil particles or

Table 1. Comparison of ambient fine and coarse mode particles. Source: EPA Paper (1996).

	FINE MODE	COARSE MODE
Formed from:	Gases	Large solids/droplets
Formed by:	Chemical reaction; nucleation; condensation; coagulation; evaporation of fog and cloud droplets in which gases have dissolved and reacted.	Mechanical disruption (e.g., crushing, grinding, abrasion of surfaces); evaporation of sprays; suspension of dusts.
Composed of:	Sulfate, SO_4^{2-} ; nitrate, NO_3^- ; ammonium, NH_4^+ ; hydrogen ion, H^+ ; elemental carbon; organic compounds (e.g., PAHs, PNAs); metals (e.g., Pb, Cd, V, Ni, Cu, Zn, Mn, Fe); particle-bound water.	Resuspended dusts (e.g., soil dust street dust); coal and oil fly ash; metal oxides of crustal elements (Si, Al, Ti, Fe); CaCO_3 , NaCl, sea salt; pollen, mold spores; plant/animal fragments; tire wear debris.
Solubility:	Largely soluble, hygroscopic and deliquescent.	Largely insoluble and non-hygroscopic.
Sources:	Combustion of coal, oil, gasoline, diesel, wood; atmospheric transformation products of NO_x , SO_2 , and organic compounds including biogenic species (e.g., terpenes); high temperature processes, smelters, steel mills, etc.	Resuspension of industrial dust and soil tracked onto roads; suspension from disturbed soil (e.g., farming, mining, unpaved roads); biological sources; construction and demolition; coal and oil combustion; ocean spray.
Lifetimes:	Days to weeks	Minutes to hours
Travel Distance:	100s to 1000s of kilometers	< 1 to 10s of kilometers

mineral ash particles, and are basic, and light in color. The accumulation mode is acidic and also oily because some of them result from photochemical reaction processes. That is why we now make a distinction between fine and coarse particles, in terms of their sources and their potential for producing adverse health effects.

REVIEW OF AMBIENT AIR QUALITY STANDARDS FOR PARTICULATE MATTER

With that background information, we can now consider how air quality measurements have been made. We have had an evolution in terms of the methods used to measure mass distributions. Initially, in the United States, we tried to measure total suspended particulate matter (TSP). We used what was essentially a big vacuum cleaner, and sucked up whatever we could onto an eight-by-ten inch filter, and used the resulting gravimetric concentration as an index of health risk.

The decision to use TSP as an index was a serious mistake, but it became the basis for the original (1971) particulate matter ambient air standard. It was a poor choice because the largest particles that were collected were too big to get into the respiratory system, and yet they could dominate the mass concentration on a windy day.

Thus, in 1987, EPA switched their measurement index to PM₁₀, which could properly be

called thoracic particles. At about 10 microns and smaller in aerodynamic diameter, you can inhale these PM₁₀ particles into the thoracic part of the respiratory tract, where they can produce some health effects.

PM₁₀ samplers use an inertial separating system to prevent the entry of the rocks as they are being sampled. In 1997, the PM ambient air standard was once again revised to include separate limits for fine particles (PM_{2.5}), since the fine particles were more closely associated with mortality and other health effects than was PM₁₀. While most of the fine particles are smaller than 1 μm, the sulfate particles, which are a major mass component of PM_{2.5} in the eastern United States are hygroscopic and grow into dilute solution droplets. If you want to capture them under humid conditions, you really have to include particles up to 2.5 μm to make sure that you captured all the hygroscopic fine particles.

There are both daily and annual average concentration limits. For the daily maxima, allowances need to be made for exceptional weather patterns. Thus, the limits are expressed as the fourth highest concentration over a three-year period, as described in Table 2. The revised PM₁₀ standards, and new PM_{2.5} standards promulgated by EPA in 1997, survived legal challenge, and were upheld by the Supreme Court.

EPA, by changing the basis for a PM₁₀ exceedence in a community, in effect reduced the number of people impacted because, in the west,

Table 2. 1997 revisions: U.S. National Ambient Air Quality Standards (NAAQS).

	1987 NAAQS	1997 NAAQS	
Index pollutant	PM ₁₀	PM ₁₀	PM _{2.5}
Annual av. concentration limit (μg/m ³)	50	50	15
Daily concentration limit (μg/m ³)	150	150	65
Basis for excessive daily concentration	4 th highest over 3 year period	>99 th percentile av. over 3 years	>98 th percentile av. over 3 years
Number of U.S. counties expected to exceed NAAQS	41	14	~150
Number of people in counties exceeding NAAQS	29x10 ⁶	~9x10 ⁶	~68x10 ⁶

SESSION III. Acid Rain Impacts: State of the Science

on windy days with high levels of soil-like particles, there were legal exceedances that were not really considered much of a health hazard.

The stringency of the PM_{10} standard was thus reduced, and the most stringent limits are now in the annual average limits of the fine particle ($PM_{2.5}$) standard. The scientific basis lies in that mortality is most closely associated with fine particles, and we now have 15 micrograms per cubic meter as the limit for the annual average in a community.

The $65 \mu\text{g}/\text{m}^3$ daily limit for $PM_{2.5}$ is a relatively relaxed standard. When the annual $PM_{2.5}$ standard is implemented, it can be expected to more than double the number of people living in areas of PM exceedence unless, of course, ambient air levels change in the meantime.

HEALTH EFFECTS OF AMBIENT AIR PARTICULATE MATTER

What is the evidence for health effects? It is

largely epidemiological associations between measured air quality parameters and mortality and morbidity indices. Figure 4, from EPA's PM Criteria Document (U.S. EPA, 1996), shows that there is a significant daily mortality risk associated with PM_{10} that is not greatly influenced by the presence of criteria pollutant gases.

Hopefully, at some time in the future we will get past reliance on these rather simple gravimetric indices of exposure to ambient air particles. Having a non-specific particle standard is hardly different in concept from having a non-specific gas standard, lumping ozone, CO, SO_2 , and NO_2 together. Unfortunately, we don't yet have the basis for deciding which components of the particle mixture are most closely associated with the health effects.

The candidate chemical components of ambient PM currently being most seriously considered as likely causal factors include: hydrogen ion content, the number concentration of ultrafines, the soluble transition elements – iron and vanadium in particular, and perhaps the

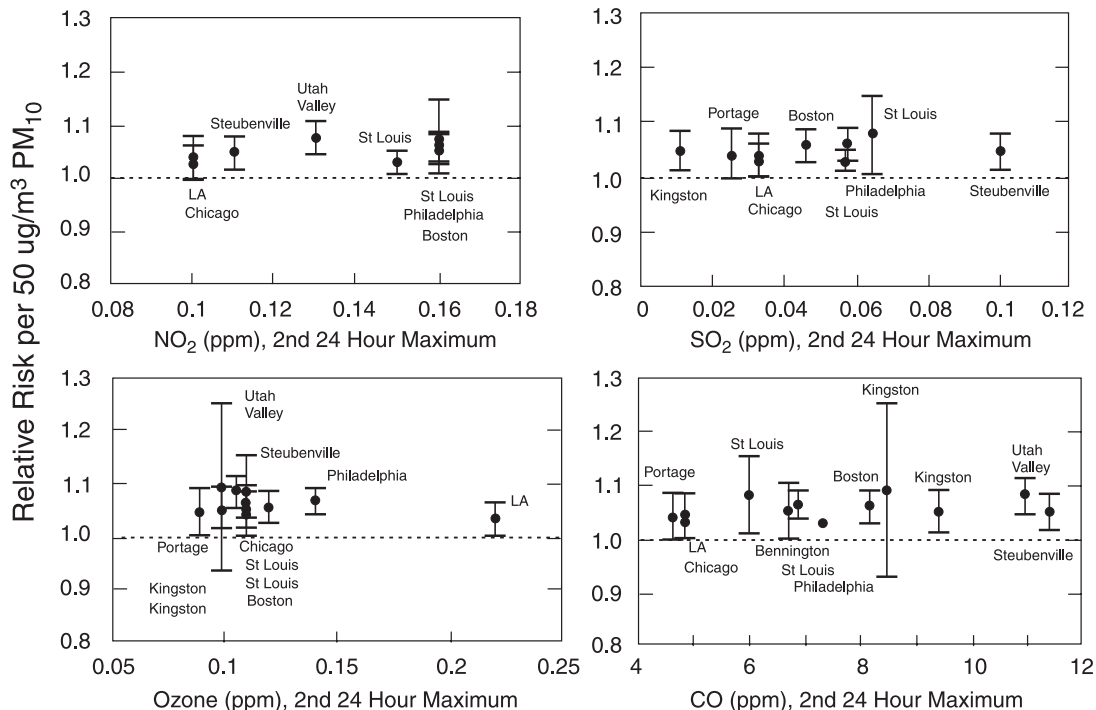


Figure 4. Relationship between RR for excess daily mortality associated with PM_{10} and peak daily levels of other criteria pollutants. (Source: U.S. EPA, 1996).

peroxides. Epidemiological evidence is, however, lacking because we don't have monitoring data for these components.

The new PM monitoring networks, i.e., the Super Sites and the speciation sites that EPA has set up in connection with the need to monitor PM_{2.5} concentrations will be providing some of the evidence that will be needed in the near future, so that the next time that an ambient air particulate matter standard comes up for review, beyond this current round, we may be able to establish a better index than fine particle mass.

In the recent years, there has been a lot of relevant health research to consider.

One of the recent landmark studies, clearly, is the Health Effects Institute (HEI) sponsored study by Samet et al. (2000), which described associations between daily excess mortality in 90 U.S. cities in relation to daily levels of PM₁₀. In this study, using standardized data on both measured PM₁₀ and mortality, there was a highly statistically significant indication of an association. This study provided further evidence than we had early in the 1997 standard setting exercise that daily mortality is more closely associated with thoracic particles than with other air criteria pollutants. This judgement was based on multiple regression analyses with the other criteria pollutants. When ozone, or SO₂, or CO, or NO₂, were included into a multiple pollutant regression, there was very little shift in the PM coefficient. This strengthens the basis for the need to regulate particles.

The Samet et al. (2000) study also examined whether there was a uniform risk coefficient around the country. They divided the U.S. data for the 90 cities into geographic regions. The increase in mortality per 10 micrograms per cubic meter increase in PM₁₀ was greater in the northeast than in the other regions of the country.

This suggests that composition does make a difference, and provided some support for the hypothesis that aerosol acidity, which is higher in concentration in the northeast than elsewhere in the U.S., may be an important risk factor. In any case, smaller, but statistically significant effects were seen in all regions of the country. It is just the magnitude that may be different in different regions, when using gravimetric mass as an index of risk.

We also have an enhanced data base on the associations between daily average PM concentrations and daily rates of hospital admissions for various respiratory diseases. The highest excess risk per unit of particles is for asthma. Pneumonia is next highest, and chronic obstructive pulmonary disease generally also shows significant excesses.

While the excess risks are small, they are coherent with the mortality effects (Bates et al., 1992), i.e., some people may be especially susceptible in some way or another, by advanced age, by disease, and are unable to cope with these inhalation challenges.

In very recent years, there has been a huge shift in research emphasis toward studies of the association of PM and cardiovascular disease. While the relative risks are highest for respiratory diseases, as might be expected for an inhaled pollutant, the total numbers of people affected appears to be greater for cardiovascular disease because there is a larger underlying prevalence of such disease in the population as a whole.

Many recent studies show associations between hospitalizations for all cardiovascular disease, and congestive heart failure in one study, heart failure and ischemic heart disease in another study.

Now, while the studies on acute effects are most abundant and highly consistent, and do tell us that there are health effects, the most stringent of the new standards is focused on the annual average concentration, which is the hardest to achieve (see Table 2).

Why is that? The cohort studies of annual mortality rates of the populations in the Harvard Six Cities (Dockery et al., 1993) and the American Cancer Society cohort (Pope et al., 1995) indicated that the mortality rates in different communities varied in relation to both fine particles (50 communities) and their sulfate content (151 communities). Their findings for sulfate and TSP are illustrated in Figure 5. It is notable that the cumulative impact of excess mortality was about three to four times greater than that indicated by summing the daily excesses from the daily mortality studies. In the economic benefit/cost analyses performed for the Clean Air Act between 1970 and 1990, 90 percent of the benefits that could be both quantitated and monetized were associated with the mortality end point, using the mortality

coefficient for $PM_{2.5}$ established for the ACS cohort (EPA, 1997). One aspect of this study was its analyses of longevity reduction. For the population average, this was in the order of one to two years. One can also look at these data in another way. For example, one can estimate that, for those more sensitive people who were affected, they may, on average, have lost about 15 years of life span (Brunekreef, 1997).

Because of the importance of their cohort mortality studies, the HEI was asked to sponsor a re-analysis of the Harvard six cities and ACS cohort studies. They did it by a completely independent team and came up with essentially the same results (HEI, 2000).

So, while EPA stuck its neck out in basing the 15 microgram $PM_{2.5}$ annual average PM limit on these two annual mortality cohort studies in establishing the $PM_{2.5}$ limits in 1997, their judgement has been confirmed by the HEI reanalysis.

FURTHER RESEARCH ON HEALTH EFFECTS OF AMBIENT AIR PARTICULATE MATTER

Further study of these cohorts is currently underway. Since they were last studied as a population, enough years have elapsed that perhaps the number of people who have died in the interim is up by a factor of about three, and we can get much more specific in terms of components and causes for that excess annual death rate.

The Harvard School of Public Health is undertaking further analysis of their Six-Cities cohort, and we, at NYU, in collaboration with Dr. C. Arden Pope III at Brigham Young University, are doing the follow up analysis of the American Cancer Society cohort.

Thus, considerable progress is being made in clarifying the relationships between exposures to airborne particulate matter, but we will need to know much more before we can optimize source controls to reduce the health and environmental impacts of both acidic sulfates and other fine particles.

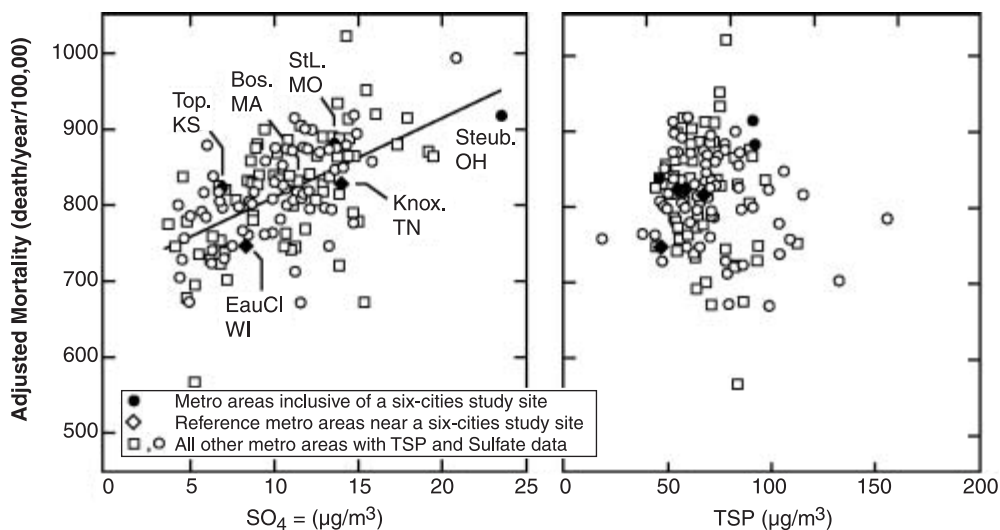


Figure 5. Age-, sex-, and race-adjusted population-based mortality rates for 1980 plotted against mean sulfate air pollution levels for 1980. Data from metropolitan areas that correspond approximately to areas used in prospective cohort analysis. (Adapted from Pope et al., 1995).

REFERENCES

Bates, D.V. 1992. Health indices of the adverse effects of air pollution: The question of coherence. *Environ. Res.* 59:336-349.

Brunekreef, B. 1997. Air pollution and life expectancy: Is there a relation? *Occup. Environ. Med.* 54:781-784.

Dockery, D.W., C.A. Pope, X.P. Xu, J.D. Spengler, J.H. Ware, M.E. Fay, B.G. Ferris, and F.E. Speizer. 1993. An association between air pollution and mortality in six U.S. cities. *N. Engl. J. Med.* 329 24:1753-1759.

Health Effects Institute. 2000. Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality. A Special Report of the Institute's Particle Epidemiology Reanalysis Project.

Ministry of Health. 1954. Mortality and Morbidity During the London Fog of December 1952. Her Majesty's Stationary Office, London.

Pope, C.A. III, M.J. Thun, M.M. Namboodiri, D.W. Dockery, J.S. Evans, F.E. Speizer, and C.V. Heath, Jr. 1995. Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. *Am. J. Respir. Crit. Care Med.* 151:669-674.

Samet, J., F. Dominici, S. Zeger, J. Schwartz, and D. Dockery. 2000. National Morbidity, Mortality, and Air Pollution Study. Part 1: Methods and Methodologic Issues. Health Effects Institute Research Report No. 94.

U.S. EPA. 1996. Review of the National Ambient Air Quality Standards for Particulate Matter: Policy Assessment of Scientific and Technical Information. OAQPS Staff Paper. Office of Air Quality Planning and Standards. EPA-452/R-96-013.

U.S. EPA. 1997. The Benefits and Costs of the Clean Air Act 1970 to 1990. EPA Report to

Congress. Office of Air and Radiation. Office of Policy. (Oct. 1997).

QUESTIONS

MR. OREN: Craig Oren, Rutgers. Do you see any indication that there is any kind of so-called threshold level of particulates that is, in some sense, safe, or does the relationship between the concentration and effects exist, even at very low concentrations of particulate matter?

MORTON LIPPMANN: The data, which are abundant, and which are quite consistent, do not indicate any threshold. Thus, EPA cannot set a standard to fully protect the public health with a margin of safety, because we, as a society, can't get down below the non-anthropogenic background of fine particles from various natural sources.

On the other hand, the available evidence doesn't prove that there isn't a threshold.

MR. SALKOVITZ: I am Dan Salkovitz, Virginia Department of Environmental Quality. I was looking at some of the data that you put up and it just occurred to me, have you done, or do you have information about the synergistic effects, for example, high fine particulate in combination with, say, ozone? What threshold levels – maybe they would be lower. I don't know. Do you have any information about that?

MORTON LIPPMANN: There are lots of data on ozone. Ozone appears to be independently associated with mortality, but to a much lesser extent than PM.

In terms of ozone and physiological measurements, the responses look linear down to background levels. So, there is no evidence of a threshold for the physiological responses to short-term ozone exposures.

When you look at some specific ozone health effects, such as hospital admissions for respiratory disease, ozone has a significant effect. In fact, if you can separate the effect of the PM and the ozone in the statistical models, ozone has a bigger effect on hospital admissions than PM does, although they both influence it. That is unlike mortality, where PM drives it, and ozone has a much smaller effect.

SESSION III. Acid Rain Impacts: State of the Science

Synergism can't be ruled out. On the other hand, the kinds of data that show that the gaseous pollutants do not materially change the coefficient for particles reduces the likelihood that it is a major factor.

MR. POIROT: Rich Poirot, Vermont air program. You had indicated that the controlling standard for the fine particle standard was the long-term annual average one, as a response to evidence for chronic effects.

The Canadian standard, I believe, is one that is based on a short-term metric. Could you comment on the difference between those two and what are some of the implications?

MR. LIPPMANN: Well, first of all, I could say that we are right and they are wrong. Seriously, the data are very hard to interpret. Different people look at it and come up with different reasons.

The Canadian reliance on the daily mortality data can be justified based on the greater amount and consistency of the database. There are more than 30 valid studies of daily mortality which are roughly consistent, and there were only two readily interpretable cohort studies for the annual mortality effect.

A major advantage of the daily mortality data is that you don't have to worry about region-to-region differences in factors like smoking, the presence of varying levels of other pollutants, the differences in ethnic composition, and dietary preferences that go with them. You are looking at the same population on a day-to-day basis, only a small fraction of whom succumb, too small to affect the next day's result.

When you are looking at multiple city results on an annual basis, all of these other risk factor considerations could be confounders that could

explain, theoretically, the mortality differences from region to region.

What made the two cohort studies so important was that they had individual data on influential factors, including diet, smoking, and occupation in the cohort data base, and could show that they didn't influence the mortality coefficients.

Also, there had been about eight cross-sectional studies of annual mortality using PM_{10} or sulfate as the index that didn't have data on individual risk factors. These previous studies were discounted because they lacked the ability to correct for these factors. In fact, they had shown the same kind of impact as the cohort studies, but people said that all these other factors could explain it. Well, they didn't.

In fact, when you consider the data from the two cohort studies that had data on the individual risk factors other than pollution, those other factors didn't explain the excess mortality. In my view, these previous cross-sectional study results gave greater credence to the results of the two cohort studies, because these two cohort studies showed that the diet, the ethnicity and so forth, even including smoking, didn't make much difference.

Therefore, the consistency of the estimated effect from the cohort studies with those produced by the prior cross sectional annual mortality studies was notable. Furthermore, the HEI re-analysis gives them a much greater weight. The re-analysis looked at the data base in every possible way and came to the same conclusion.

I can understand the reluctance of other authorities to rely more on the annual mortality data, but I think they are wrong.