Background on Health Effects of Acid Aerosols

by Morton Lippmann*

This introduction to the 1987 NIEHS-EPA Symposium on the Health Effects of Acid Aerosols reviews the state of our knowledge on this topic as of the close of the 1984 NIEHS Conference on the Health Effects of Acid Precipitation (Environmental Health Perspectives, Volume 63) and the results of some key studies completed since that time. These studies, together with the results of the studies presented in the papers that follow, provide a substantial increment in our knowledge of the health effects of acid aerosols.

Introduction

This Symposium on the Health Effects of Acid Aerosols is taking place 3 years after the NIEHS Conference on Health Effects of Acid Precipitation. The earlier conference, also convened at the NIEHS, covered the effects of acid precipitation on mobilization, transport, and speciation of toxic metals, the impact of acid precipitation on metals toxicity, and the health effects of inhaled acid aerosols. Its proceedings were published in Environmental Health Perspectives, Volume 63. This symposium was organized to help the sponsors (NIEHS and EPA), the larger scientific community, and Congress address and assess the expanding data base on the effects of inhaled acid aerosols.

To the extent that we can place the emerging exposure and effects data into a broader perspective on the public health consequences of current and future exposures, we can help the sponsors identify data gaps, research needs, and the possible need for a primary national ambient air quality standard for acid aerosols. To this end, we depend on the skills and insights of the rapporteurs of our half-day sessions on Exposure, Epidemiology, Toxicology, and Acute Human Responses to present integrations and interpretations of the presentations in their sessions. We will also benefit from an evaluation of the potential uses and limitations of the current data base on acidic aerosols for risk assessment by Roy E. Albert (I). His broad perspectives, combined with his experience in experimental studies on the effects of inhaled irritants, environmental epidemiology, and quantitative risk assessment, enable him to evaluate the data base from a broad perspective.

Historical Background

Industrial pollution from steel mills and sulfuric acid (H₂SO₄) plants were implicated in excess deaths and respiratory illness among elderly people in The Meuse Valley in Belgium in December 1930, and in Donora, PA, in October 1948. Coal smoke containing H₂SO₄ was implicated in excess deaths, primarily in elderly people in a series of episodes in London from 1952 to 1962, with approximately 4000 excess deaths from the December 1952 episode alone. During that period, deaths attributable to chronic bronchitis were higher in London than in other, less polluted areas in the U.K., and much higher than in other countries in northern Europe. Aggravation of bronchitis among patients in London with chronic lung disease was also demonstrated to occur as pollution levels increased. Unfortunately, levels of exposure to H₂SO₄ were not determined in the era when excessive mortality and morbidity were clearly evident, and exposure-response relationships were made, if at all, with pollutant indices referred to as British Smoke (BS) and sulfur dioxide (SO₂). The actual measures were the blackness of sampled particles (BS) and the net acidity of sampled vapor (SO₂).

It is now clear that excess mortality in London was much more closely associated with BS than with SO₂. However, BS is primarily a measure of the carbon content of the aerosol, an unlikely causal factor. It seems much more likely that another aerosol constituent, e.g., the free hydrogen ion (H⁺), was a causal factor.

Many of the reasons for the interest in and concern about H⁺ or H₂SO₄ as one dominant source of ambient H⁺, were discussed at the 1984 NIEHS conference. My assignment at that conference was rapporteur for the session on the health effects of inhaled acid aerosols. Table 1 from the proceedings of that conference
indicates the questions I had formulated prior to the conference. It also presents the answers I could come up with by the end of the conference, the conditions under which they were applicable, and the remaining knowledge gaps at that time. My responses were based, in large measure, on material presented at the conference, but were supplemented, as appropriate, with knowledge of other relevant work (2).

Recent Progress

Three years is not a long time for progress in research on so broad and complex an issue as the health effects of acid aerosols, especially when there have been so few active investigators and resources available to address the knowledge gaps. Most of the active research groups are represented at this symposium and will be presenting their recent work to us. It is therefore too early to fully gauge the progress that has been made, although much of it is significant and moves our knowledge base well past that of 3 years ago, as summarized in Table 1. Most of the recent research provides exposure-response data involving directly measured acid aerosol exposures. However, we are not yet at the stage where we can afford to ignore studies with surrogate measures of exposure. Reports from Germany and Poland at this symposium are good examples of papers of great interest that still rely on surrogate metrics of exposure.

Other recent work not being reported at this sym-

| Question | Answer* | Applicable conditions | Knowledge gap |
|----------|---------|-----------------------|---------------|
| Have health effects from exposure to ambient acid aerosols been demonstrated in the past? | Yes | ~600 cases morbidity; primary H2SO4; point source avg. exposure > 160 µg/m³ | Diagnostic criteria; exposure variability in time and space |
| Do current North American exposures to ambient acid aerosols produce measurable health effects? | Uncertain, but likely | Asthma admissions, secondary aerosol | H⁺ vs. surrogate SO₄²⁻ |
| What are current North American exposures to ambient acid aerosols? | Max. ~30 µg/m³ as H₂SO₄ (6 hr avg.) | Summer, rural N.E. U.S. | Spatial, diurnal, and seasonal variations |
| What are the effects of single brief exposures to acid aerosols on respiratory mechanics? | Transient bronchoconstriction in exercising asthmatics | 100 µg/m³ H₂SO₄, adolescents 350 µg/m³ H₂SO₄, adults | Effects in more severely disabled patients |
| What are the effects of single brief exposures to acid aerosols on particle clearance from the lungs? | Persistent reduction in DLCO in guinea pigs | < 40 µg/m³ H₂SO₄ | Significance to human health effects |
| What are the effects of repetitive exposures to acid aerosols on lung structure and function? | ↑ mucociliary clearance | ≤ 100 µg/m³ H₂SO₄, humans | Effects on lung epithelium |
| | ↑ alveolar clearance | ≤ 250 µg/m³ H₂SO₄, rabbits | |
| | ↑ activation of macrophages | > 750 µg/m³ H₂SO₄, rabbits | |
| What are the implications of persistent structural and functional alterations in the lung to the pathogenesis of chronic respiratory disease? | T-B tree: unknown, of concern Repetitive daily exposure to irritants, stimulation of mucus secretion | Continuous mild inflammatory response | Effects on respiratory mechanics and symptoms at later stages |
| | Alveolar region: unknown, of concern because persistent inflammation can lead to abnormal repair processes | | Effects of macrophage activation and protracted recruitment of inflammatory cells and fibroblasts; structural changes in deep lung |
| Are there especially sensitive subgroups in the population? If so, who are they? | Yes: asthmatics, especially adolescents and with vigorous exercise | Bronchoconstriction, 100 µg/m³ H₂SO₄ or 0.3 ppm NO₂ | Effects in more severely disabled patients |
| Do other ambient pollutants potentiate the effects of acid aerosols on the respiratory tract? | Yes | | Other mixtures, sequences of exposures |

*↑ an acceleration in rate; ↓ a retardation in rate.
Table 2. Growth-adjusted pulmonary function differences in 7- to 11-year-old school children during and after an air pollution episode in IJmond, the Netherlands, January 16–20, 1985 (3).

| Lung function parameter | January 18 (n = 62) | February 6 (n = 60) | February 15 (n = 41) |
|--------------------------|---------------------|---------------------|---------------------|
| FVC, mL                  | −62 (± 11)*         | −58 (± 13)*         | −2 (± 16)           |
| FEV0.5, mL               | −39 (± 9)*          | −30 (± 13)*         | −5 (± 13)           |
| FEV1, mL                 | −50 (± 10)*         | −43 (± 13)*         | 7 (± 15)            |
| PEF, mL/s                | −219 (± 62)*        | −69 (± 76)          | −162 (± 68)*        |
| MEF25%, mL/sec           | −100 (± 67)*        | −50 (± 70)          | −36 (± 58)          |
| MEF50%, mL/sec           | −37 (± 44)          | −70 (± 48)          | 7 (± 47)            |
| MEF75%, mL/sec           | −27 (± 24)          | −62 (± 34)          | 26 (± 38)           |
| MMEF, mL/sec             | −38 (± 32)          | −60 (± 44)*         | 12 (± 40)           |

*−p < 0.01 (one-sided).
'−p < 0.05 (one-sided).
''−p < 0.1 (one-sided).

Ozkaynak and Thurston's results from regression model for 98 U.S. SMSAs and for 38 SMSA subset with directly measured FP and IP concentrations using the 1980 total annual mortality and ambient particle data.*

| Coefficient (SE) for pollution variable, deaths/10^5 persons per year per μg/m^3 | 98 SMSA | 38 SMSA |
|-----------------------------------------------------------------------------------|---------|---------|
| SO2                                | 6.6 (1.5)† | 6.9 (2.1)* |
| FP                                 | 2.2 (0.8)† | 2.8 (1.4)* |
| IP                                 | 0.3 (0.3)  | 0.3 (0.5)  |
| TSP                                | 0.1 (0.2)  | 0.1 (0.3)  |
| IP                                 | 2.9 (2.1)  | 1.0 (0.9)  |

*Model includes the variables percent over 65 years, median age, log of population density, percent with 4 years of college, and percent below poverty line.
†Estimates of FP based on regional FP equations developed by Ozkaynak and Thurston (4).
*Estimates of IP based on approach developed by Trijonis (6).
*−p < 0.05.
†p < 0.01.
‡p < 0.001.

Figure 1. Plot of total mortality rate versus annual mean sulfate concentration (left panel) and total suspended particulate matter (TSP) concentration (right panel) in the 98-SMSA subset. (Letters are initials of states.) Adapted from Ozkaynak and Thurston (4).
Metropolitan Statistical Areas (SMSAs) and four measures of particulate air pollution. These were total suspended particulate matter (TSP); inhalable particulate matter, i.e., particulate < 15 μm in aerodynamic median diameter (IP); fine particulate matter, i.e., particulate < 2.5 μm in aerodynamic median diameter (FP); and sulfate (SO$_4^{2-}$), a major component of FP. This was a final report on the preliminary analyses reported by Ozkaynak and Spengler (5) at the 1984 NIEHS Conference. They found that FP and SO$_4^{2-}$ were most consistently and significantly associated with the reported SMSA-specific total annual mortality rates, whereas TSP and IP were often nonsignificant predictors of mortality. These results are summarized in Table 3 and Figure 1. Figure 2 is my adaptation of the results of the SMSA-mortality analyses, showing a hypothetical extrapolation of the analytical results based upon the hypothesis that the acid component of the particulate, a greater fraction as one goes from TSP to IP to FP to SO$_4^{2-}$, is the causal factor. The results reported at this symposium provide a basis for examining the validity of this hypothesis, and/or for helping to establish a better one.

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REFERENCES

1. Albert, R.E. Risk assessment for acid aerosols. Environ. Health Perspect. 79: 201-202 (1988).
2. Lippmann, M. Airborne acidity: Estimates of exposure and human health effects. Environ. Health Perspect. 63: 63-70 (1985).
3. Dassen, W., Brunekreef, B., Hoek, G., Hofschreuder, P., Staatsen, B., deGroot, H., Schouten, E., and Biersteker, K. Decline in children’s pulmonary function during an air pollution episode. J. Air Pollut. Control Assoc. 36: 1223–1227 (1986).
4. Ozkaynak, H., and Thurston, G. D. Associations between 1980 U.S. mortality rates and alternative measures of airborne particle concentration. Risk Anal. 7: 449-461 (1987).
5. Ozkaynak, H., and Spengler, J. D. Analysis of health effects resulting from population exposures to acid precipitation precursors. Environ. Health Perspect. 63: 45-55 (1985).
6. Trjonis, J. Development and application of methods for estimating inhalable and fine particle concentrations from routine hi-vol data. Atm. Environ. 17: 999-1008 (1983).