

The Annapolis Center For Science-Based Public Policy



"Our greatest responsibility is to be good ancestors."
...Jonas Salk

A Critique of the Campaign Against Coal-Fired Power Plants



PREFACE

Several years ago, The Annapolis Center published a report on potential health effects of particulate matter (PM)*. That report found the science used to justify the new air quality standard for fine particulate matter (PM_{2.5}) was weak at best. In the attempt to justify changing the air quality standard for particulate matter from PM₁₀ to PM_{2.5}, the report noted:

Some epidemiologic studies link current levels of ambient air pollution to statistical increase in the number of deaths per day. An association between particulate air pollution and mortality is also suggested by the pattern of findings in these studies as the results are examined in total. Other studies also suggest that measures of illness or morbidity, such as the numbers of hospitalizations among the elderly, are affected by PM. Scientists usually seek to complete their understanding of such observational evidence by performing toxicological studies to identify the mechanisms of injury. However, past studies focused on concentrations and particle sizes that are not directly relevant to today's lower levels of exposure and did not elucidate what it is about PM that might be contributing to observed health effects even at the higher concentrations used in the experiments. That gap in our knowledge constitutes a key uncertainty in interpreting the observational data. It is the interpretation of epidemiological studies that has been at the center of the current debate. The debate continues because the toxicologic and mechanistic understanding of PM is absent.

This present paper, which was written by an expert in air quality health effects and reviewed by physicians and other scientists, adds to the Center's work on the use of epidemiology in decision-making while focusing on a public relations campaign against coal-fired power plants and the health effects of PM. In our view further study of this subject is warranted by a responsible expert, unbiased organization such as the National Academy of Sciences. For scientists and lay-people alike, we hope that it explains our concerns about this campaign.

Sincerely,

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Former Surgeon General, United States Navy
Chair and President,
The Annapolis Center for Science-Based Public Policy

* "The PM Report: A Discussion of the Science for the U.S. EPA's Proposed Particulate Matter Rule" (1997)

A Critique of the Campaign Against Coal-Fired Power Plants

In the last several years, several technical reports and press releases have claimed that particulate matter (PM) from coal-fired power plant emissions is responsible for widespread human health problems in the United States.

These reports were sponsored by an environmental advocacy group (the Clean Air Task Force, or CATF) and a charitable foundation (the Pew Charitable Trust). Members of the Harvard School of Public Health provided part of the technical leadership. Only one of these reports has been published in the peer-reviewed scientific literature. Much of the more recent peer-reviewed research seems to call the conclusions of many of these reports into question.

“A Critique of the Campaign Against Coal-Fired Power Plants”, commissioned by The Annapolis Center¹, provides a synopsis of the approach, assumptions and conclusions of the CATF reports and contrasts them with the results derived from the peer-reviewed scientific literature. This comparison shows that CATF’s claims of a linkage between currently operating power plants in the U.S. and community ill health lack scientific support. This comparison was undertaken because the Annapolis Center for Science-Based Public Policy is concerned about the quality of science used in public-policy decision-making.

The main air pollutants emitted by fossil-fueled power plants are sulfur dioxide (SO₂), oxides of nitrogen and NO_x, and particulate matter (PM), for which strict emission limits and ambient air quality standards have long been established. Additional PM may be formed downwind as a product of SO₂ and/or NO₂ reactions in the atmosphere, as sulfate or nitrate compounds, usually as various salts of ammonia.

Since reaching their peak in the 1970s, total power plant emissions of SO₂ have dropped by about one-third. Since the 1960s, ambient atmospheric concentrations of SO₂ have dropped by over 90 percent. Concentrations of atmospheric particulate sulfates have also declined over this period; particulate nitrate concentrations are not routinely monitored. Ambient concentrations of NO₂ have dropped by more than half since the 1960s, mainly in response to emission controls on vehicles.

In addition to demanding that SO₂ emissions from power plants be reduced even further, the CATF also demands reductions in the emissions of oxides of nitrogen. A National Ambient Air Quality Standard (NAAQS) exists only for NO₂.

Particles emitted from power plants vary in size and composition. Fly-ash particles from coal tend to be glassy spheres, high in alumina-silicates but also contain carbon and several trace

¹ “A Critique of the Campaign Against Coal-Fired Power Plants” is available through The Annapolis Center at www.annapoliscenter.org

elements. Oil fly-ash particles tend to be sooty agglomerates high in sulfur and trace metals. The ambient standards for PM do not distinguish differences in the chemical composition of PM (neither does most of the extant epidemiology). Most of the current violations of the ambient PM standards are thought to result from fugitive dust. (Fugitive dust refers to emissions from roadways, material handling, agriculture, etc.) The official EPA measures of PM changed in 1987 from "total suspended particulates" (TSP) to "PM₁₀" (particles captured by a filtering device designed for particles mainly less than 10 microns in diameter [about 10 percent of the width of a human hair]), making long-term trend comparisons more difficult. PM₁₀ concentrations have declined about 2-3 percent per year since 1989. In 1996, EPA promulgated a new ambient standard for even smaller particles: PM_{2.5} or "fine" particles. The available ambient data also indicate decreasing PM_{2.5} concentrations over time, even in rural areas.

The first report from the CATF was that of Levy *et al.* (1999), which dealt with two power plants in the Greater Boston area and was partly based on an earlier peer-reviewed publication² and Levy's doctoral thesis at Harvard. A similar report dealt with nine coal/oil-fired plants in Illinois. "Death, Disease, and Dirty Power" and the technical report from Abt Associates upon which it was based were published in the fall of 2000. Finally, CATF released "Power to Kill" in July 2001. This document attributed health impacts to 51 older power plants that have been accused of making mechanical modifications so that they would be re-classified as "new" rather than "existing" sources and thus be required to conform to tighter emission standards. This is the "New Source Review" (NSR) issue.

The non-peer-reviewed reports attempt to make the point that their estimated health impacts would constitute an intolerable public health burden. However, there are large differences in the mortality estimates among the six reports. The non-peer reviewed analysis of two large coal-fired plants claimed there were twice as many deaths per ton of SO₂ emission and an order of magnitude more deaths per MW of generating capacity than was reported in Levy *et al.* (1999), even though the meteorology and population distributions should have been the same as in the peer-reviewed report. CATF claims that plants located on the East Coast would have health impacts similar to those located in the interior of the country are not credible, given the generally westerly winds that prevail and the higher population density typical of the Eastern Seaboard.

The reports in the CATF campaign did not follow the standard risk assessment paradigm, instead choosing to use a "damage function" approach, in which selected epidemiological findings from the literature are adapted to the problem at hand, regardless of the situations under which they were derived. Accepted methods of risk assessment involve several separate and sequential steps³:

- Hazard assessment (what specific compounds are toxic?)
- What should the dose-response functions look like (are thresholds to be expected?)

² Levy JI, Hammitt JK, Yanagisawa Y, Spengler JD, Development of a new damage function model for power plants: Methodology and applications, Environ Sci Tech 33:4364-72 (1999).

³ National Research Council of the National Academy of Sciences, Risk Assessment in the Federal Government: Managing the Process, National Academy Press, 1983.

- Exposure assessment (who is exposed to these compounds and at what levels?)
- Risk characterization (integration of all of the above to provide an assessment of the risks of specific hazards for specific populations).

One of the most prominent deficiencies of the CATF analyses is the surprising assumption that all particles that might be collected by a mass sampler (including fugitive dust) are equally responsible for a panoply of health effects, ranging from heart attacks to upper respiratory infections. A few consider effects of ammonium nitrate (NH_4NO_3); none of them were referenced by CATF. The toxicology literature clearly exonerates ammonium sulfate and nitrate at the ambient concentrations in question. It is thus inappropriate to frame all of the health impacts considered here in terms of the mass concentrations of $\text{PM}_{2.5}$ or PM_{10} . This was recently emphasized in an EPA toxicology paper⁴, which stated: "...mass may not be the most appropriate metric to use in assessing health effects after PM exposure but rather specific compounds must be identified and assessed."

A further shortcoming of the CATF reports is that they do not take into consideration the potential negative health effects associated with their recommended pollution control measures. The accepted protocol for risk analyses and for formal documents like environmental impact statements require that offsetting factors be considered. Thus, the CATF analyses do not meet normal scientific standards and the CATF model should be regarded as only a theoretical construct. (In accord with the Annapolis Center's recently published report "*Epidemiology: In Decision-Making*", a "weight of evidence" approach is necessary, in which information from all relevant disciplines is brought to bear in assessing causality.)

The critique shows the following with respect to the CATF/Harvard conclusions and its supporting information:

1. Methods used by CATF to predict air quality impacts from these plants have not been validated by comparisons to actual measurements. Results from two alternative models that were used differ by an average of 60 percent; the extent to which either of them represents reality is unknown.
2. Only one of the papers and reports in the CATF campaign was peer-reviewed (Levy *et al.*, 1999). That report based its estimates of PM-related health effects on acute effects and its health effect estimates were lower than those in the other reports by an order of magnitude.
3. The CATF health effects estimates are not based on the specific compounds emitted from coal-fired power plants or on compounds that are formed from them downwind in the atmosphere. Instead, they are based on the assumption that all constituents of PM_{10} are equally toxic, a premise that is without support.

⁴ Ghio AJ, Devlin RB, Inflammatory lung injury after bronchial instillation of air pollution particles, *Am J Respir Crit Care Med* 164:704-8 (2001).

4. Results from recent peer-reviewed epidemiology studies run counter to the epidemiology studies upon which the CATF/Harvard estimates are based. A recent EPA-sponsored epidemiological study⁵ of long-term effects on mortality found “no evidence that the observed relationships of PM_{2.5} with mortality could be accounted for by confounding by SO₂ or SO₄ (sic).” Given the methodological flaws in the CATF studies, their findings are thus not an appropriate basis for setting pollution control policy.
5. With respect to ambient PM, airborne sulfates are the most important power plant-related pollutants. There is no support in the experimental or occupational health literature for the adverse health effects that are attributed to these compounds at present ambient concentrations. This conclusion also holds for ammonium nitrate.

Our conclusion is that the CATF/Harvard claims of ill health caused by current power plant emissions of sulfates and nitrates in the U.S. lack the scientific support required for use in developing regulatory policy.

⁵ McDonnell WF, Nishino-Ishikawa N, Petersen FF, Chen LH, Abbey DE, Relationships of mortality with the fine and coarse fractions of long-term ambient PM₁₀ concentrations in nonsmokers, J Expos Anal Environ Epidemiol 10: 427-36 (2000).

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A Critique of the Campaign Against Coal-Fired Power Plants

Introduction

In the last several years, technical reports and press releases appeared that attempted to link emissions from coal-fired power plants with widespread human health effects throughout the United States.¹⁻⁵ By and large, these plants have been operated within legal emission limits, and the ambient air quality in their vicinities continues to meet the applicable standards for sulfur dioxide (SO₂) and nitrogen dioxide (NO₂), the two main pollutants that are emitted from properly controlled coal combustion. These reports were sponsored by an environmental advocacy group (the Clean Air Task Force) and a charitable foundation (the Pew Charitable Trust). Some members of the Harvard School of Public Health provided part of the technical leadership. Since this work was performed, much new research has been published that calls its basic assumptions into question.

The purpose of this critique is to contrast the approach and assumptions used by the Clean Air Task Force (CATF) with the facts that are readily available from the published, peer-reviewed literature. This comparison shows that CATF's accusations of linkage between power plants and ill health are without scientific support. Their approach is based on application of epidemiological findings from the literature, dating back a decade or more, as opposed to a bona fide risk assessment. Further examination of the literature on air pollution and health suggests that the electric power industry should be encouraged to produce more power, not less, and under the most efficient operating conditions possible. Improved efficiency is consistent with the desire to reduce CO₂ emissions and will make air conditioning more affordable. Use of residential air conditioning is an effective public health measure, since it protects against heat-waves^a and greatly decreases exposures to outdoor air pollutants and allergens.

The critique begins by considering CATF's claims in the context of historical patterns in U.S. emissions and air quality. The currently accepted methodology for risk assessment is discussed, together with an examination of the substances emitted by coal-fired power plants, their toxicity, and their relative importance compared with other sources (hazard assessment). Air quality models are considered briefly, and the CATF dose-response functions are assessed by examining the relevant long-term epidemiology, including recent studies and the extent to which the epidemiology is supported by other types of health studies (see Appendices). This critique concludes that the CATF claims lack the scientific support required for use in developing regulatory policy.

a. Rogot et al.⁶ found that in hot weather, the mortality rate for persons who had central air conditioning was 42% lower than for persons without central air, after controlling for confounding variables.

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The CATF Publications and Their Conclusions

The first report to surface from this campaign was apparently that of Levy et al. (2000)¹, which dealt with two power plants in the Greater Boston area. This analysis was based on the model of Levy et al. (1999),⁷ with a receptor grid extending over an area of about 700 by 500 km. The pollutants considered included SO₂, NO₂, PM₁₀, SO₄²⁻, NO₃-, and O₃. Health effects considered included premature mortality and 11 different indices of morbidity; mortality accounted for the bulk of the imputed economic impacts. A further application of the Levy et al. model was for the effects of 9 coal/oil fired plants in Illinois over an area of about 900 x 600 km covering portions of nine midwestern states.² This report states that wet and dry deposition were included in the model for these applications, which was apparently not the case in the Massachusetts study.¹

"Death, Disease, and Dirty Power"³ and the technical report from Abt Associates upon which it was based⁴ covered the entire United States but used different air quality modeling methods. "Fine" particles were emphasized, as formed secondarily from SO₂ and NO_x emissions. Health endpoints included premature mortality and six morbidity indices.

Finally, "Power to Kill"⁵ appeared in July 2001 and emphasized impacts attributed to 51 older power plants that have been accused of making sufficient mechanical modifications to classify them as "new" rather than "existing" sources and thus to require conformance with tighter emission standards. This is the "New Source Review" (NSR) issue. The report also emphasized fine particles but the discussion of health impacts was limited to premature mortality and asthma attacks.

All of these publications except the one that was peer-reviewed for publication⁷ make the point that their estimated health impacts constitute an intolerable public health burden. Table 1 compares the results of the various analyses in terms of "excess" deaths per MW of electrical capacity or kT of SO₂ emitted. "Deaths per unit of SO₂" reflects the propensity of most of these analyses to emphasize secondary sulfate particles as the most toxic agent; "deaths per MW" would include all other pollutants as well.

Table 1 Excess Mortality Indices from Various CATF Publications

Number of Plants	Reference	Location	Capacity (MW)	SO ₂ emission (000T/y)	SO ₂ /MW (tons/MW)	Deaths Per Year	Deaths Per MW	Deaths Per 000T SO ₂ /y
1	6	MA	57	0.33	6	0.31	0.005	0.93
2	1	MA	2,416	75	50	159	0.066	2.1
8	5	VA	~13,000	650	50	1,930	0.15	3.0
9	2	IL	7,596	151	20	400	0.053	2.65
51	5	AL,GA,IL, FL,KY,OH, TN,NC,SC, MS,WV,IN	66,000	3,568	54	~5,650	0.086	1.58
All	4	All US	~400,000	13,217	33	30,100	0.075	2.27

There are some surprising differences in the mortality indices among these six cases. The one peer-reviewed publication⁷ shows by far the lowest indices, because this is a cogeneration plant with very low SO₂ emissions in relation to NO_x emissions and the electricity generated is only part of its output, the others being steam and chilled water. The authors noted that most of the excess mortality was attributed to ozone; possible chronic effects of PM on mortality were not considered in the baseline estimates. By contrast, the (non-peer reviewed) analysis of two large coal-fired plants¹ in the same metropolitan area showed twice as many deaths per unit of SO₂ emission and an order of magnitude more deaths per MW of capacity, even though the meteorology and population distributions should have been the same. It is also surprising that plants located on the East Coast should have impacts similar to those located in the interior of the country, given the generally westerly prevailing winds. Also, if the national study⁴ had used the PM-mortality relationship of the peer-reviewed study,⁷ the estimated death toll would have been reduced by an order of magnitude, to around 0.1% of annual U.S. mortality. Finally, note that the 51 plants with the highest SO₂ emissions per MW of installed capacity have next to the lowest mortality ratio per unit of SO₂ emission. This suggests that those plants, which apparently use higher sulfur coals than the national average (by far), must be located rather far from major population centers.

Calculations of this type are not new, having been featured in government energy planning scenarios in the late 1970s. Rowe⁷ estimated population exposures to primary PM from power plants for the entire country on a unit-emission basis, using a Lagrangian long-range transport model. These estimates varied by two orders of magnitude across the nation, depending on meteorology and population density. The population-weighted median exposure was 0.0005 ug/m³ per kT of PM emission. Based on the 1998 estimate of 273 kT of primary PM emissions from U.S. coal-fired plants, the population-weighted impact of primary PM emissions from coal-fired power plants would be 0.14 ug/m³. This is less than 1% of EPA's new ambient PM_{2.5} standard. Using the (acute) mortality risk factor of Levy et al.⁷ (5×10^{-6} annual deaths/person/ug/m³) and a population of 280 million results in an estimate of 191 deaths/y for the entire nation (less than 1/100th of a percent). Rowe also published similar calculations for sulfates,⁹ but only for the eastern half of the country and based on summer meteorological conditions. Here the unit exposures were somewhat lower (a median figure of 0.00029 ug/m³ per kT of SO₂ was estimated from the published contour map), but of course, SO₂ emissions are much higher. Using an estimated current annual emission rate of 10 million tons for coal-fired plants in this part of the country yields a median ambient SO₄²⁻ concentration estimate of 2.9 ug/m³, which is somewhat lower than current measurements would indicate, suggesting other sources of ambient SO₄²⁻.

The Basic CATF Analytical Framework and Its Assumptions

CATF's basic methodology involves the superposition of effects from multiple sources and thus requires linear dose-response functions (also known as "damage" or "concentration-response" functions [CRFs] with no thresholds. CATF considered selected sources of uncertainty using randomization (Monte Carlo) methods, but the most important questions, that of the validity of the underlying air quality and health impact models, are not addressed. Measured ambient air quality did not enter into this process,

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and the long-term health damage functions have never been validated through epidemiological studies of actual changes over time in response to pollutant abatement. Thus, the entire CATF risk analysis framework should be regarded as a theoretical construct.

The accepted basic methods of risk assessment involve several separate and sequential steps:¹⁰

1. Hazard assessment (what specific compounds are toxic?)^b
2. What should the dose-response functions look like (are thresholds to be expected?)?
3. Exposure assessment (who is exposed to these compounds and at what levels?)
4. Risk characterization (integration of all of the above to provide an assessment of the risks of specific hazards for specific populations).

By contrast, the CATF approach ignores the first two steps, in favor of the "damage function" approach, in which selected epidemiological findings from the literature are adapted to the problem at hand, regardless of the situations under which they were derived. A further major shortcoming of the CATF reports is the total neglect of the disbenefits of the pollution control measures that are recommended. Such compensating effects should have been considered in a balanced analysis and include:

1. Impacts of global warming due to increased CO₂ emissions resulting from loss in efficiency from imposition of pollution controls.
2. Increases in retail electricity prices that would:
 - a. reduce the use of air conditioning with consequent health impacts.
 - b. cause shifts to more polluting forms of space heating, especially wood burning.
 - c. cause dislocations of businesses with concomitant loss of jobs.
3. Effects of market disruptions and increased prices for natural gas resulting from utilities shifting from coal to gas.
4. Unanticipated changes in ambient air quality arising from the complex nonlinear atmospheric chemistry, including:
 - a. local increases in ambient ozone because of reductions in NO.
 - b. increases in particulate nitrate due to reductions in SO₂.
5. Costs and environmental impacts of disposing additional scrubber sludge.

The accepted protocols for risk analyses and for formal documents like environmental impact statements require offsetting factors to be considered. Thus, the CATF analyses do not meet these standards.

- b. Air pollution research and policy distinguishes various types of particles for emphasis. EPA policy dictates that only pollutants of outdoor origin are to be regulated under the Clean Air Act. Toxicological research tends to focus on carbon black (soot) and metals. Ghio and Devlin¹¹ concluded that "mass may not be the most appropriate metric to use in assessing health effects after PM exposure but rather specific components must be identified and assessed.

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Assessment of Health Hazards from Coal Combustion Emissions

The first step in an assessment of health risks from power plant air pollution should be a detailed consideration of the hazards of each compound or class of compounds that is emitted. Briefly, those compounds are sulfur dioxide (SO_2), oxides of nitrogen (NO_x , consisting of NO and NO_2), carbon dioxide (CO_2), and fly ash. Under normal operating conditions, utility boilers do not emit appreciable amounts of either carbon monoxide (CO) or unburned hydrocarbons (VOCs). Trends in the national averages of major air pollutants may be obtained from Table 2 (1997 was the latest year considered in the project that produced this table¹²). Note that the numbers of counties with air quality data have expanded considerably since the 1960s, which may have had the effect of overstating some trends, since the early data were mainly obtained in the larger cities. Figure 1 shows the trend in measured ambient SO_4^{2-} values, computed as the average of all available stations (Table 2 averages over each county before computing the annual average). The substantial difference between data obtained from high-volume (TSP) samplers that use glass-fiber filters and the size-classified dichotomous samplers is due to artifact SO_4^{2-} formed on the alkaline TSP filters from SO_2 in the air being sampled.¹³ This difference is also seen in Table 2. Note that SO_4^{2-} is not a "criteria" (Federally regulated) pollutant, and thus the availability of ambient data is largely a matter of state options. Data for years after about 1982 are thus sparse; hence the use of "estimated" values based on empirical relationships.¹² Most of the power plants in contention by CATF came on line in the 1960s and 1970s; these increased emissions thus do not seem to have greatly affected ambient air quality for sulfur oxides.

Sulfur dioxide has been the main focus of CATF, in part because power plants are the major source of SO_2 in the United States and because sulfur compounds have been identified with noxious air pollution since medieval times¹⁴. Since the 1960s, ambient SO_2 has dropped by over 90%, even though emissions have only dropped by about one-third since their peak in the 1970s¹⁵ and many new large power plants have come on line. This reduction in ambient SO_2 was accomplished mainly by using cleaner fuels for space heating, since emissions from ground level sources have much more effect on the air that people breathe than do emissions from the tall stacks that are typical of power plants. There were no violations of ambient standards for SO_2 in 1998 (the most recent year for which EPA reported data¹⁶); the worst county in the U.S. met the annual standard by a margin of 30%.

CATF also demands reductions in the emissions of NO_x from power plants. Unlike SO_2 , there are many diverse sources of NO_x since these compounds are formed in all combustion processes, with the rates of formation highly dependent on flame temperature.¹⁷ The primary compound emitted directly from combustion is nitric oxide (NO), which is then oxidized to nitrogen dioxide (NO_2) through atmospheric reactions with ozone (O_3). Ambient standards are limited to NO_2 , for which national averages have dropped by more than half since the 1960s (Table 2). National emissions of NO_x have been stable since about 1980, at about 65% higher than they were in 1960.¹⁵ Since 1980, ambient levels have dropped by about 25%, apparently because the shares of (relatively constant) national emissions have shifted from 37% attributed to on-road vehicles and 26% for coal-fired power plants in 1980, to 32% for on-road vehicles and 30% for coal-

fired power plants in 1994.¹⁵ CATF includes atmospheric nitrates (presumably as ammonium nitrate) in its estimates of power-plant contributions to ambient PM_{2.5}; however, there is no medical evidence supporting long-term health effects due to inhaling nitrates (see Appendix B).

Table 2 Trends in U.S. ambient air quality
(annual means for counties unless otherwise noted)

Period	Species	Mean	Std Dev	Number of Counties
Particulate Matter (ug/m ³)				
1960-64	TSP	96.92	41.81	281
1970-74	TSP	69.65	28.37	1,258
1979-81	TSP	60.51	19.63	1,277
1989-91	TSP	48.78	16.28	532
1979-84	PM ₁₅	38.30	10.73	101
1995-97	PM ₁₀	28.64	8.05	648
1999	PM ₁₀	23.26	6.73	675
1979-84	PM _{2.5}	19.19	5.66	101
1999	PM _{2.5}	12.97	3.74	540
Sulfate Aerosol (ug/m ³)				
1960-64	SO ₄ ²⁻ #	9.92	5.36	193
1970-74	SO ₄ ²⁻ #	9.31	3.77	294
1979-81	SO ₄ ²⁻ #	9.07	3.77	329
1982-88	SO ₄ ²⁻ #	7.34	3.88	216
1982-88	SO ₄ ²⁻ *	7.55	3.39	425
1989-96	SO ₄ ²⁻ *	5.85	3.18	425
1979-84	SO ₄ ²⁻ +	5.26	2.70	79
#glassfiber filters *estimated values +Teflon filters				
Sulfur Dioxide (ppb)				
1960-69	SO ₂	56.63	48.42	42
1970-74	SO ₂	16.18	14.62	279
1979-81	SO ₂	9.04	5.65	480
1989-91	SO ₂	7.20	4.13	411
1995-97	SO ₂	4.94	2.69	393

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Table 2 Trends in U.S. ambient air quality (continued)

Period	Species	Mean	Std Dev	Number of Counties
95 th percentile carbon monoxide (ppm)				
1960-69	CO	13.81	8.47	44
1970-74	CO	9.64	5.63	206
1979-81	CO	5.90	3.54	272
1989-91	CO	2.69	1.22	246
1995-97	CO	1.72	0.76	261
nitrogen dioxide (ppb)				
1960-69	NO ₂	35.76	11.22	33
1970-74	NO ₂	28.11	12.62	151
1979-81	NO ₂	20.06	10.22	236
1989-91	NO ₂	18.02	8.43	194
1995-97	NO ₂	15.43	7.35	240
95 th percentile ozone (ppb)				
1970-74	O ₃	146.99	51.63	156
1979-81	O ₃	120.12	36.30	452
1989-91	O ₃	84.71	14.77	439
1995-97	O ₃	73.18	10.01	520

Direct emissions of particulate matter (PM) from power plants are highly dependent upon the types of fuel and the control devices used. Coal is probably the dirtiest type of fossil fuel, comprising around 10% ash by weight, depending on its type and source. Utility-sized coal-fired boilers have long been equipped with "cyclones" that collect the largest particles through centrifugal action, more-or-less as "bottom ash." The major reduction in PM emissions from coal-fired power plants that has been achieved since 1960 (88%) has been achieved through the use of highly efficient PM collection devices (mainly electrostatic precipitators (ESPs) that trap the smallest particles, called "fly-ash". It is noteworthy that this major reduction in PM emissions from power plants has been achieved in the face of a 250% increase in power production since the 1960s and a 5% increase in the energy share produced from coal.

Like all airborne particulate matter, PM emissions from power plants are highly variable in terms of size and composition. Fly-ash particles from coal tend to be glassy spheres, high in alumina-silicates but also containing various trace elements including carbon. Oil fly-ash particles tend to be sooty agglomerates that are high in sulfur and trace metals. PM from natural gas firing is negligible. The ambient standards for PM do not distinguish between types of particles (neither does most of the extant epidemiology), and most of the current violations are due to fugitive dust. The official EPA measures of ambient PM changed in 1987 from "total suspended particulates" (TSP) to "PM₁₀"

(particles captured by a filtering device designed for particles mainly less than 10 microns in diameter [10% of the width of a human hair]), making it difficult to examine long-term trends. However, Table 2 shows a decrease of 50% in average TSP concentrations from the 1960s to 1990, and a further 15% drop in PM_{10} after that (based on the conventional PM_{10} /TSP ratio of 0.55), again, despite major increases in electricity produced from coal.

EPA considers PM emissions in two major categories: [1] combustion (3.4%), industrial processing (3.2%), and transportation (2.2%), totaling 8.8%; [2] the remaining, largely uncontrollable 91%, comprising agriculture, forestry, and wildfires (26%), and fugitive dust (65%). This inability to control the largest sources of PM_{10} was part of the rationale for setting a new standard for "fine" particles ($PM_{2.5}$), which are dominated by particles arising from combustion processes and are arguably more amenable to controls. In 1998, coal-fired power plants contributed only 9.7% of the PM_{10} emissions in the first (traditionally inventoried) category and 0.86% of all estimated PM_{10} emissions. More recently, EPA has also estimated national $PM_{2.5}$ emissions, totaling about 9.5 million tons, or about 27% of total PM_{10} emissions.¹⁸ 70% of these direct emissions of $PM_{2.5}$ are from categories that are difficult to control, including fugitive dust, agriculture, wind erosion, and off-road vehicles. The largest combustion category is for biomass burning (about 30% of the non-fugitive dust $PM_{2.5}$). Primary emissions from coal-fired power plants are only about 3% of $PM_{2.5}$ emissions, including fugitive dust, or about 5% without considering $PM_{2.5}$ fugitive dust.

However, there are other concerns about PM emissions from utilities, in part because they process such large quantities of fuel. One category is that of air toxics, compounds or elements that may be especially toxic, even in minute concentrations. EPA conducted a special study of this problem in the 1990s, involving a list of 189 potential candidates, and devoting a special effort to electric utilities.¹⁹ The only candidates for which health concerns might be warranted were nickel compounds from oil-firing, arsenic, and mercury (Hg) in coal. The latter has become a cause celebre in its own right, even though airborne Hg is a global problem because of its long atmospheric residence time and the U.S. contributes only a small fraction of that global burden. Hg exposures (through consumption of contaminated fish) are far below levels of health concerns; EPA uses a "safety factor" of 10 in setting exposure limits to partially bridge this gap and to heighten public awareness.

There is a substantial difference between the ratio of ambient $PM_{2.5}$ to ambient PM_{10} (0.55) and the corresponding ratio of direct emissions (0.27). The difference is due to secondary PM formed in the atmosphere, comprising sulfate compounds (SO_4^{2-}) from SO_2 , nitrate compounds (NO_3^-) from NO_x , and organic carbon particles formed from volatile organic carbon gases. East of the Mississippi, SO_2 comprises about 1/2 to 2/3 of rural $PM_{2.5}$ ¹⁶ and nitrate is mainly found only in winter. Nitrate is more important in the West, comprising up to 1/4 of rural $PM_{2.5}$ and more in some areas of Southern California. Since about 2/3 of the SO_2 is emitted by power plants, sulfates have become the largest PM species associated with these sources and thus are the main focus of the CATF campaign. For this reason, it is important to examine the various types of evidence linking human health effects with sulfur compounds.

CATF's Bases for Estimating Health Effects Resulting from Coal-Fired Power Plant Emissions

Air Quality Models. The CATF/Harvard studies predicted exposures from various types of air quality models, depending upon the situation. None of these model predictions were verified against ground truth. The PM concentrations attributed to power plants were based on oxidation of SO₂ to sulfuric acid (H₂SO₄) that is then neutralized by ambient ammonia (NH₃) to form ammonium sulfate ([NH₄]₂SO₄) and oxidation of NO_x to form nitric acid (HNO₃) that is also neutralized to become ammonium nitrate (NH₄NO₃). These compounds are thus jointly considered as PM_{2.5}, without distinction, and their harmful effects are assumed to result from the combined mass of PM, not its chemical composition. The various models use different assumptions for their gas-phase and aqueous oxidation rates as well as for dry and wet deposition rates, which become critically important in long-range transport, such as on the national scale.³⁻⁵ These models apparently do not consider the scenarios proposed by West et al.,²⁰ in which reduced sulfate levels lead to increased availability of NH₃ to react with gaseous nitric acid to form additional particulate nitrate.

The Abt analyses considered two different national air quality-modeling schemes, whose results differed by a ratio of about 1.6 for the Eastern U.S.⁵ and up to 2.4 in more distal locations. Only one set of results was used in the other two reports,^{3,4} presumably the higher ones. The maximum PM_{2.5} increment attributed to power plants is 7 ug/m³, occurring in the vicinity of Tennessee.⁴ If this increment were all due to sulfates, the concentration would be about 5 ug/m³ in SO₄²⁻ units, which is somewhat lower than ambient values measured there ca. 1980 (SO₄²⁻ = 8.1, PM_{2.5} = 20.8).²¹ These ambient measurements show that about half of the PM_{2.5} in the region consists of compounds other than SO₄²⁻ and that a good portion of the measured sulfate must have come from sources other than power plants. These comparisons do not support the validity of the Abt models and underlying assumptions.

Dose-response Functions. The CATF³⁻⁵ and Harvard^{1,2,7} reports vary somewhat in terms of the bases used to predict health effects, all of which were taken from the extant literature, 1987-1999. Levy et al.⁷ considered all the criteria pollutants, but based their central estimates only on acute mortality, relegating the much larger estimates of chronic mortality effects to "sensitivity" studies. Unfortunately, the smaller estimates were based on a value of \$6.1 million per statistical life, notwithstanding the small reductions in life expectancy typical of daily mortality relationships.^c Levy et al.⁷ included variations in mortality value in the sensitivity studies, resulting in order-of-magnitude decreases.

c. Relative risks attributed to daily PM exposure are of the order of 1.005 to 1.02 per 10 ug/m³. Using the conventional formula that relates risk to age at death, these risks correspond to losses in life expectancy of 3 weeks to 3 months. This same formula predicts a loss of 8 years due to smoking, for example. A totally different approach developed by Murray and Nelson²² based on estimates for the frailest portion of the population predicts a loss due to air pollution of only 2-3 days. Thus, there are major differences.

All of the other (non-peer reviewed) CATF reports used various estimates from the American Cancer Society study²³ for the long-term mortality effects of PM_{2.5}, having converted them to a PM₁₀ basis. This epidemiology study is discussed in more detail below..

One of the most glaring deficiencies of the CATF analyses is thus the failure to recognize the specific compounds for which they are demanding controls: atmospheric sulfates, mainly ammonium sulfate ($[\text{NH}_4]_2\text{SO}_4$). There are many epidemiological studies in the literature that specifically consider SO₄ as a potentially toxic agent.²⁴ By electing to frame all of the health impacts considered here in terms of PM_{2.5} or PM₁₀, CATF implicitly assumes that all particles that might be collected by a mass sampler (including fugitive dust) are equally responsible for an entire panoply of health effects, ranging from heart attacks to upper respiratory infections, which is counterintuitive. More specific information on the health effects of sulfates is given in Appendix A.

The American Cancer Society Study of Long-Term Mortality and Air Pollution (ACS)

Study Design. In the mid-1990s, two epidemiology studies were published that broke new ground with respect to long-term associations between air pollution and mortality. The Harvard Six Cities Study²¹ used research-grade air quality data in conjunction with relative rates of survival among volunteers in six locations; this study found about 25% lower survival in the most polluted city, but could not definitively attribute the excess mortality to a specific pollutant. This study was followed by an epidemiology study that used data from the American Cancer Society's Cancer Prevention Study II (CPS-II)²³ along with air quality data from routine surveillance monitors, but only for PM_{2.5} and sulfate. About 1.2 million U.S. adults were enrolled by Society volunteers in 1982. These participants are more likely than the general U.S. population to be college-educated, married, middle-class, and white. Unlike the Harvard Six Cities Study (H6CS),²¹ ACS was not originally intended to deal with potential risks of ambient air pollution, although the enrollment questionnaire sought information on industrial exposures to various chemicals. No clinical data were obtained during the ACS study.

ACS was intended to validate H6CS and thus was designed to follow more-or-less the same protocol. As a result, it did not take advantage of the richer database afforded by CPS-II and was restricted to the two air pollutants that had been singled out in H6CS: PM_{2.5} and SO₄. Air quality data ca. 1979-81 were obtained from two different sources: for PM_{2.5}, from EPA's Inhalable Particulate Network (IPN), as reported by Lipfert et al.,²⁵ the SO₄²⁻ data were apparently obtained indirectly from AIRS through Harvard and other sources. Unfortunately, these two air quality networks differed greatly in geographic coverage and in the types of filters used: IPN used largely nonreactive teflon filters, but the SO₄²⁻ data were obtained from reactive glass-fiber filters that tend to inflate the SO₄²⁻ readings.¹³ The independent variables used in ACS were the same as in H6CS, with the addition of alcohol consumption. The geographic unit that was selected for cross-sectional analysis was the Standard Metropolitan Statistical Area (SMSA). SMSAs are groups of contiguous counties [except in New England] surrounding a major city, and sometimes, multiple cities.

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In contrast with H6CS, Cox proportional hazards models were estimated with air pollution (either $\text{PM}_{2.5}$ or SO_4^{2-}) entered as a continuous independent variable. Because there were 50 locations with $\text{PM}_{2.5}$ data and 151 with SO_4^{2-} data, two different models were necessarily estimated. No information was given in ACS as to how well the non-pollutant estimates compared between models. H6CS used the same mortality model with all pollutants.

The authors of H6CS were careful not to claim that air pollution actually initiates chronic disease, only that "air pollution contributes to excess mortality." Such a conclusion is consistent with the reported results of many of the time-series studies and by the episode experiences of past decades and thus is not really at issue. However, ACS went further stating, "combustion source air pollutants may be important contributing factors **causing** respiratory illness and early mortality due to cardiopulmonary diseases" (emphasis added). This conclusion is much less defensible because the pollutants in question had not been specifically linked to combustion sources and because no linkage has been established with respiratory illness per se. Exposures that contribute to the etiology of a disease that ultimately kills must occur decades before death; such lagged air quality data were not considered in ACS (or H6CS).

Many technical questions were raised by these two studies, especially when EPA decided that they provided evidence of the need for an annual $\text{PM}_{2.5}$ ambient standard. A key issue was the refusal of the authors to make the basic data available to others for reanalysis. A compromise was reached when the Health Effects Institute (HEI) contracted with a Canadian group (Krewski et al.) to reanalyze the data and to perform sensitivity studies, with strong guidance from the original investigators (OIs). This reanalysis report was released in the summer of 2000²⁷ and some of its findings were used in the CATF reports. Respiratory mortality was not mentioned by CATF; neither study found significant increases to be associated with air quality, and ACS found a negative relationship between sulfate and respiratory mortality.

Air Quality Data Used by the Original Investigators (OIs) Table G-5 in Appendix G of Part II (Sensitivity Analyses)²⁷ lists air quality data used for ACS (the data for H6CS were listed in the original paper); this is the first such listing and it raises several questions. Checking the fine particle data entries reveals some facts that were not included in the original publication. It appears that whoever extracted the $\text{PM}_{2.5}$ data from the Brookhaven National Laboratory (BNL) report²⁵ was under the (mistaken) impression that the listings were for SMSAs, when, in fact they are for cities. This should have been obvious from the fact that analyses in the BNL report were done for up to 960 locations, whereas there were only about 300 SMSAs at the time. As a result, the city of Los Angeles was taken to represent the Los Angeles SMSA without including the city of Pasadena in the SMSA average, and $\text{PM}_{2.5}$ for the SMSA was taken as 21.81 rather than as 26.44 $\mu\text{g}/\text{m}^3$. Similar errors were made for Minneapolis-St. Paul (only Minneapolis was used) and for Houston (Seabrook, TX, was ignored).

During the early stages of the Reanalysis project, HEI and the reanalysis contractors were notified of the availability of an updated (and presumably official) version of the IPN

data from EPA, which they eventually obtained. This version included more locations and a slightly longer period of time. It does not appear that the newer IPN data are listed in Appendix G and it is thus not possible to confirm if SMSA assignments were made properly. However, the highest PM_{2.5} value in the updated database, 42 ug/m³ for San Bernadino, CA, is not listed in Table G-5.

The sulfate data used in ACS are of unknown provenance. The OIs used data from 151 SMSAs; the reanalysis could confirm data for only 144 of them. The reanalysis project obtained all of their supplementary air quality data from an independent contractor, not directly from EPA or any other official source; thus, all of the ACS SO₄²⁻ results must be considered as unverified.

Lagged Exposures. Both ACS and H6CS used ambient air quality data roughly coincident with the periods of follow-up. In a steady-state situation, this would have made little difference, and the gradients among cities could have been assumed to also pertain to prior periods corresponding to the initiation of the chronic diseases that eventually killed some of the participants. However, the periods in question, 1976-1987 and 1982-1989 were preceded by periods of great change in ambient air quality in the United States, so that this specific situation is decidedly not steady-state. In general, dirty cities got much cleaner while initially clean cities either stayed the same or deteriorated slightly due to population growth. Thus, the range of variation within the dataset has decreased by about a factor of 4-5, which should have led to a similar reduction in the PM regression coefficients, relative to lagged data. Now, the equivalent PM_{2.5} regression coefficient (in percent per ug/m³) as calculated by the original authors would be about 6.4% per 10 ug/m³, which is considerable larger than typical values from time-series studies. However, if the lagged PM values had been used instead, the cross-sectional regression coefficient would be reduced to about 1-2% per 10 ug/m³ (assuming that the older data fit the mortality ratios equally well), which is not statistically different from the acute values in the literature. The conclusion follows that accounting for disease latency would drastically change the interpretation of the effects in the context of current air quality and the likelihood of chronic responses. This conclusion also applies to the estimates made by CATF, which were based on PM_{2.5}.

Ecological and Other Confounding Covariates. Krewski et al.²⁷ (Appendix Table E-1 of Part II, Sensitivity Analyses) list candidate ecological variables that were considered and the reasons for inclusion or exclusion, as well as data sources used for those that were included. Most of the reasons for exclusion involve putative lack of data, but data on demography, climate, and benzo(a)pyrene (a carcinogenic PM species) are in fact readily available from standard sources. New England was omitted from the models incorporating population change because SMSA data were not available; however county data could have been used instead. Further, the underlying ACS database includes data on diet, exercise, and subjects' heights, none of which was used in the final ACS models. It thus it appears that the sensitivity studies of Krewski et al. stopped short of evaluating all of the potentially important confounding variables. It has been shown that cross-sectional studies such as ACS involving regionally distributed data are highly prone to overstating the effects [rather it's more the aspect of overstating the statistical

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significance] of similarly distributed pollution variables (such as $\text{PM}_{2.5}$ and SO_4^{2-}), because of spatial autocorrelation.^{25, 28}

Summary Discussion. The H6CS and ACS studies differ in important ways. H6CS has better air quality data and subject follow-up but too few locations to allow the effects of collinear pollutants to be separated. ACS has many more subjects and locations and thus statistical power but has been based on air quality data of unknown reliability. Further, actual exposures are less certain in ACS because of averaging over SMSAs and because changes in residential locations were not tracked during follow-up.

The two studies agree in that they both find concordant spatial patterns in air pollution and in mortality that are controlled by higher levels of both parameters in the industrial Midwest and Appalachia, as have many ecological studies before them. However, this regional concordance and the resulting statistical relationships are unique in the United States and perhaps worldwide.

What has the HEI Reanalysis Project²⁷ accomplished?

1. It has shown that the published results^{21,23} were not the results of accidental errors or inappropriate statistical methods, within the constraints of the models posited by the OIs and the data that they elected to use. However, until the air quality data used by ACS have been validated, especially for SO_4^{2-} , these results should be considered tentative, even within the constraints of the OI models.
2. The reanalysis has revealed details of the studies that were not available in the original papers, including the inappropriate coding of missing data on alcohol use and questions about the air quality data used in ACS.
3. Whereas the original publications combined cardiac and respiratory deaths together, the reanalysis also treated them separately and showed negative associations with respiratory deaths. The combined category was positive because there are typically many more cardiac than respiratory deaths.
4. The reanalysis showed that significant mortality effects were limited to persons of low education; it was unable to discern possible reasons for this, but incomplete treatment of socioeconomic status in the regression model is one possibility.
5. The reanalysis showed that (gaseous) SO_2 is a better predictor of excess mortality in the ACS study than either sulfate or $\text{PM}_{2.5}$, even though many fewer people are actually exposed to SO_2 than to SO_4^{2-} . The lower exposure to SO_2 results from its tendency to be absorbed onto interior surfaces, such that air concentrations are much lower indoors (where adults spend ca. 90% of their time) than outdoors. The finding that SO_2 predicts mortality nevertheless is a strong indicator that both SO_2 and SO_4^{2-} are actually geographic indicator variables instead of long-term health risk factors per se.

6. Sensitivity studies show many instances in which the pollution effects could be diminished through modeling and/or data changes. There were no instances of important increases in the pollution coefficients by means of such changes. One might thus conclude that the original estimates are likely to be over-estimates.
7. The reanalysis showed that spatial autocorrelation was indeed present in the ACS study, and that accounting for it tended to reduce the magnitude of the pollution estimates and to widen their confidence limits.
8. Altogether, the reanalysis shows that, in considering long-term mortality responses to air pollution, there are many important issues to be considered other than those included in the OIs' simple model. Krewski²⁷ recognized this in his final paragraph (p.234): "Finally, it is important to bear in mind that the results of our reanalysis alone are insufficient to identify causal relations with mortality."

What problems still remain?

1. The report does not address why significant negative responses to NO₂ and (mean) O₃ should be given any less credibility than significant positive findings to SO₂, SO₄²⁻, and PM_{2.5}.
2. It did not investigate the effects of individual subjects' stature (height), of peak (as opposed to mean) ozone, of lagged exposures, of the metal content of PM, of known carcinogenic air pollutants (such as benzo(a)pyrene), or of measured personal exposures to SO₄²⁻ and PM_{2.5}.²⁹
3. It has not shown whether the mortality associations are robust to the use of SMSAs instead of cities or counties or to the inclusion/exclusion of certain locations. Indeed, it seems possible that quite different results might be achieved by abandoning the OIs' modeling approach and starting from scratch with a model that takes advantage of all of the ACS and exogenous data known to be available.
4. The reanalysis provides no basis for judging as to the time scale of response, i.e., as to whether the associations represent responses to long-standing pollution levels over years or decades or simply the accumulation of much shorter (acute) responses.
5. It has not shown which pollutants might be the most important or which sources of those species should be further controlled.
6. The full range of available ecological variables was not explored.
7. The true effects of population change on the pollution coefficients were masked because data for New England were not used, where some substantial population losses have occurred.
8. The reanalysis provided no clues as to which of the many alternative pollution estimates would be the most credible for policy purposes.

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9. Although "semi-individual" prospective cohort studies are undoubtedly more acceptable to the epidemiology community than purely ecological designs, the reanalysis fails to show any overwhelming advantages for the former. The ACS study was shown to exhibit significant spatial autocorrelation when models were restricted to individual-level variables alone, thus requiring that the design become "more ecological" in order to achieve valid significance tests.
10. The reanalysis does not support coherence with the time-series studies, in that responses were stronger for younger subjects and for those without prior disease, as well as failing to confirm the relationships with CO, NO₂, and O₃ that have been found in many acute mortality studies.

In summary, most of the earlier criticisms of ACS and H6CS²⁶ remain unaddressed. The H6CS and ACS studies have been used in cost-benefit studies here (such as the CATF reports) and abroad and have been interpreted as showing that air pollution causes new cases of chronic disease, in addition to shortening the lives of already-compromised individuals. However, this interpretation largely rests on the magnitudes of the putative mortality responses, in comparison to those seen in time-series studies of daily mortality. Limited as it was in the use of ecological variables, and notwithstanding the neglect of other individual variables such as height and diet, the reanalysis shows that, when more complete models are used, these cross-sectional long-term estimates overlap the acute estimates. This would likely have been even more apparent had lagged exposures been used in the long-term studies. Note that use of a value of \$6 million for loss of life that is unlikely to exceed a few weeks is not justified, and thus that the economic ramifications of the CATF studies are also problematic. Until these remaining modeling- and data-related questions are resolved, the results of H6CS and ACS should not be used for policy purposes.

Relevant Findings from Additional Recent Long-Term Mortality Studies

While the literature on acute health effects of air pollution is voluminous, only a few studies deal specifically with PM_{2.5} or sulfates, and in general, those findings are inconsistent.^{24,51} Since 5 of the 6 CATF-related reports emphasize long-term health effects and since the bulk of the claimed economic effects stem from long-term mortality impacts, we turn to the recent literature on long-term health effects to examine the extent of support for CATF's claims.

The EPRI-Washington University Veterans' Cohort Mortality Study.³⁰ This paper presents the design and some results from a new prospective mortality study of a national cohort of about 50,000 U.S. veterans who were diagnosed as hypertensive in the mid-1970s, after approximately 24 years of follow-up. This national cohort is male with an average age at recruitment of 51 +/- 12 y; 35% were black and 81% had been smokers at one time. Because the subjects have been receiving care at various U.S. Veterans Administration (VA) hospitals, access to care and quality of care are relatively homogeneous. Non-pollution predictor variables in the baseline model include race, smoking (ever or at recruitment), age, systolic and diastolic blood pressure (BP), and

body mass index (BMI). Interactions of BP and BMI with age were also considered. Although this study essentially controls for socioeconomic status by design because of the homogeneity of the cohort, selected ecological variables were also considered at the zip code and county levels, some of which were found to be significant predictors. Pollutants were averaged by year and county for TSP, PM₁₀, CO, O₃, and NO₂; SO₂ and Pb were considered less thoroughly. Both mean and peak levels were considered for gases. SO₄²⁻ data from the AIRS database and PM_{2.5}, coarse particles, PM₁₅, and SO₄²⁻ from EPA's Inhalable Particulate (IP) Network were also considered. Four relevant exposure periods were defined: 1974 and earlier (back to 1953 for TSP), 1975-81, 1982-88, and 1989-96. Deaths during each of the three most recent exposure periods were considered separately, yielding up to 12 combinations of exposure and mortality periods for each pollutant. Associations between concurrent air quality and mortality periods were considered "acute," responses associated with prior exposure were considered "chronic," and pre-exposure mortality associations were considered to be indirect (non-causal).

The implied mortality risks of long-term exposure to air pollution were found to be sensitive to the details of the regression model, the time period of exposure, the locations included and to the inclusion of ecological as well as personal variables. Statistically significant mortality responses were found in both directions, i.e., adverse and beneficial. Among the positive responses, indications of acute mortality risks were seen for NO₂ and peak O₃, with an indication of chronic risks only for NO₂. The mean levels of these excess risks were in the range of 5-9%; peak O₃ was dominant in 2-pollutant models. However, the finding of even larger significant negative risks (i.e., beneficial effects of air pollution) suggests that, either the analysis is very sensitive to the deletion of locations because of missing data, or that the model specification may still be incomplete. The study showed that the response to a given pollutant, say O₃, varied during the 24-y follow-up period, suggesting the presence of a response threshold or perhaps depletion of the cohort of its most susceptible members over time. This finding shows that integrating responses over the entire period of follow-up (as was done in the Six Cities²¹ and ACS²³ studies) can be misleading. Fine particles as measured in the 1979-84 EPA Inhalable Particulate Network indicated no significant excess (positive) mortality risk for this cohort. In essence then, the conclusions of this study are diametrically opposed to those of the previous prospective cohort studies.^{21,23}

An Ecological Study of US Mortality by County, 1960-97.¹² The objective of this study was to investigate longitudinal and spatial relationships between ambient air pollution and age-specific mortality aggregated over U.S. counties. Cross-sectional regression analysis was performed for five specific periods, based on published data on mortality, air quality, demography, climate, socioeconomic status, lifestyle, and diet. The outcome measures were statistical relationships between ambient air quality and county mortality rates by age group for all causes of death, less AIDS and trauma. A specific regression model was developed for each period and age group. Criteria for including a variable in each model were statistical significance ($p < 0.05$), absence of substantial multicollinearity (variance inflation factor < 2), and the expected algebraic sign. To maximize data set size, these models were initially developed without the air pollution variables. Regression residuals were then regressed against each air quality measure in

turn, including those from previous periods, and dose-response plots were constructed. The validity of this 2-stage procedure was shown by comparing a subset of results with those obtained by including air quality in the full regression model (correlation = 0.88).

On the basis of attributable risks (computed for overall mean concentrations), the strongest associations were found in the earlier periods; responses to most pollutants declined over time. Thresholds were suggested for TSP at about 100 $\mu\text{g}/\text{m}^3$ for mean TSP, 7-10 $\mu\text{g}/\text{m}^3$ for mean SO_4^{2-} , 10-15 ppm for peak (95th percentile) CO, 20-40 ppb for mean SO_2 . Contrary to expectations, associations were often stronger for the younger age groups (<65 y). For all pollutants except sulfate aerosol, monitoring coverage has improved over time; however, the more recent periods tend to show weaker relationships, with attributable risks less than 5% in most instances. Nevertheless, stronger relationships were often seen when the locations considered were limited to conform to those of previous cohort studies. The pollution-mortality relationships were usually strongest when both parameters were measured in the same period. In the earliest period, before widespread pollution controls were implemented, strong effects were seen for all pollutants except ozone, for which data were lacking. In the 1970s and early 1980s, sulfate and SO_2 were among the most important pollutants, but these relationships decreased dramatically in more recent years. In the early and mid-1990s, peak O_3 showed statistically significant regression slopes for certain age groups, but exhibited counterintuitive (non-monotonic) dose-response relationships. $\text{PM}_{2.5}$ was significant for most age groups in 1979-81 and 1989-91, but only for ages 45-64 in 1995-97. The effects of CO, NO_2 , and traffic (measured as vehicle-miles traveled per mi^2) were mainly negative in recent periods, sometimes significantly so. When comparable periods, age groups, and pollutants were examined, reasonable agreement was found with the various published prospective cohort studies. Since this is an ecological study of relationships among the characteristics of counties and their changes over time, drawing conclusions about the relative survival rates of individuals may not be appropriate. However, on the basis of these county-level results, spatially derived relationships between air quality and mortality vary significantly by age group and period and may be sensitive to the locations included in the analysis. Further, it appears that effect thresholds were present in the early periods and, as air quality improved in the later periods, fewer counties were subjected to air pollution levels above these implied thresholds, thus weakening the overall statistical relationships with mortality. The results of this study thus indicate that thresholds are present in long-term relationships between air quality and mortality, that the relationships differ significantly by age at death, and that relationships found in past decades cannot be extrapolated to the future.

A Comprehensive Study of U.S. Infant Mortality.³¹ This paper used U.S. linked birth and death records to explore possible ecological and environmental relationships with infant mortality. The analysis considers a range of infant mortality endpoints, regression models, and environmental and socioeconomic variables. The basic analysis involves logistic regression modeling of individuals; the cohort comprises all infants born in the United States in 1990 for which the required data are available from the matched birth and death records. These individual data include sex, race, month of birth, and birth weight of the infant and personal data on the mother, including age, smoking and education in most instances, and adequacy of prenatal care. Ecological variables from

Census and other sources are matched on the county of usual residence and include ambient air quality, elevation above sea level, climate, numbers of physicians per capita, median income, racial and ethnic distribution, unemployment, and population density. The air quality variables considered were 1990 annual averages of PM₁₀, CO, SO₂, SO₄²⁻, and "non-sulfate PM₁₀" (obtained by subtracting the estimated sulfate mass from PM₁₀). Because all variables were not available for all counties (especially maternal smoking), it was necessary to consider various subsets of the total cohort. For comparable modeling assumptions, the results for PM₁₀ agreed with previously published estimates; however, these associations were not robust to changes in the model and/or the locations considered. Significant negative mortality associations were found for SO₄²⁻, and the associations with PM₁₀ were not specific to probable exposures or causes of death. There was no indication of a role for outdoor PM_{2.5}, but possible contributions from indoor air pollution sources cannot be ruled out, given higher SIDS rates in winter, in the north and west, and outside of large cities. This study showed that previous EPA claims³² that fine particles in the outdoor ambient might be responsible for certain types of infant mortality are fallacious.

The AHSMOG Studies³³⁻³⁸ Ambient air quality exposures for O₃, TSP, PM₁₀, SO₂, NO₂, and SO₄²⁻ were estimated by zip code for the period 1973-1993 for 6338 nonsmoking Seventh-Day Adventists in California. Exposures were interpolated to subjects' zip codes for work and home and averaged over time to estimate cumulative exposures. PM₁₀ was estimated from TSP for years prior to 1987. Health follow-up was from 1977 to 1992 and a 3-year lag was assumed for lung cancer incidence, for which there were 16 new male cases and 20 new female cases.³³ After adjustment for education, current alcohol consumption, and pack-years of past smoking, significant excess lung cancer risks were seen only in males, for O₃, PM₁₀, and SO₂, but not NO₂. Multiple-pollutant analyses suggested that the associations with PM₁₀ and SO₂ were independent of other pollutants. Other potential confounders (exercise, diet, time spent outdoors, occupational exposure) changed the O₃ coefficient by less than 10% and thus were not included (confounding of other pollutants was apparently not considered). The 1999 mortality study³⁴ reported significant mortality relationships with PM₁₀ for males, but not females, and for respiratory but not cardiovascular causes. PM₁₀ exposures above 100 ug/m³ were statistically significant, while mean PM₁₀ was not, implying a non-linear or threshold relationship. The earlier AHSMOG papers³⁵⁻³⁷ found no effects on mortality^{35,36}, an association with high levels of TSP or estimated mean PM_{2.5} for incidence of all cancers in females³⁶, and a marginally significant association between O₃ > 0.1 ppm and incidence of lung cancer in both genders³⁷. Sulfate was not a significant predictor in the mortality study³⁴ and was not considered in the lung cancer study.³³

In the most recent AHSMOG paper³⁸, involving research sponsored by EPA, survival in a subset (n=3769) of the cohort that lived near any of 9 California airports was considered from 1977-92 in terms of various lifestyle and personal characteristics and exposure to air pollution. Daily PM_{2.5} concentrations were estimated from airport visibility data (R=0.72), back to 1966. Daily PM₁₀ values were either measured or estimated from TSP; daily coarse particle (CP) values were obtained as the difference between PM₁₀ and PM_{2.5}. Although there were no statistically significant relationships when males and females were considered separately, for males, the relative mortality risks were

consistently higher for $PM_{2.5}$ than for CP, and the authors concluded that the PM_{10} - mortality relationship in a previous paper³⁴ was likely due to $PM_{2.5}$ rather than CP. Of the gaseous pollutants, (mean) ozone was significantly associated with deaths for which nonmalignant respiratory disease (NRD) was a factor, and the authors concluded that O_3 was likely more important than $PM_{2.5}$ for these deaths. Special attention was given to the potential role of SO_2 and SO_4^{2-} in these relationships by running 2-pollutant models with $PM_{2.5}$. For all-natural-cause mortality (ANC), $PM_{2.5}$ was by far the stronger predictor with a relative risk (RR) of 1.33. By contrast, the RR for SO_4^{2-} was only 1.06. For respiratory mortality (NRD), “no independent positive association with either SO_2 or SO_4^{2-} was observed.” Meaningful analyses of lung cancer mortality were precluded because of the small number of deaths (7).

In comparing the latest AHSMOG findings with those of the Six Cities Study²¹, McDonnell et al.³⁸ noted that both studies showed similar risks for $PM_{2.5}$, but differed markedly in their findings for sulfate, which was highly significant in the Six Cities Study²¹ but not in AHSMOG. They pointed out that SO_4^{2-} and $PM_{2.5}$ were highly correlated in the six cities ($R=0.89$) but only weakly correlated in AHSMOG ($R=0.33$). (This correlation was 0.73 in the ACS study²³ and 0.75 in the Veterans’ Study³⁰.) It thus follows that the appearance of a strong effect on mortality of SO_4^{2-} in the Six Cities and ACS studies must be due to its collinearity with $PM_{2.5}$ rather than as an independent relationship.

For most pollutants in the AHSMOG studies, the contrast is between the South Coast Air Basin (SCAB) and less polluted areas of California such as San Diego or the Bay Area, with only about 10% of the subjects in other counties. However, particulate exposures prior to 1973 were up to twice as high in Los Angeles as they were after 1973, while they were only about 25% higher elsewhere in California in the earlier period. The contrast in oxidants was also greater in earlier years, and EPA data on SO_4^{2-} and TSP that go back the mid-1950s were not used. By ignoring these earlier data, the long-term contrast in exposures appears to have been greatly underestimated in AHSMOG. Further, a latency period of three years is much too short for development of respiratory disease, especially for lung cancer. With respect to all-cause and cardiovascular mortality, CO should have also been considered as a predictor. Average CO in Los Angeles in 1963 was over 11 ppm, while CO in San Diego and San Francisco was less than half that. By 1981, all of these values were down by 2/3 or more. There were similar trends for SO_2 in SCAB, and the finding of significant long-term effects of SO_2 at levels of a few ppb is not credible, given the ready adsorption of SO_2 on indoor structural surfaces (thus further attenuating actual exposures) and in body fluids (thus preventing long-term buildup of particles).

In general, AHSMOG exposures have been based on individual addresses and concentrations interpolated from several monitoring stations. The latest paper³⁸ used airport visibility, which is by definition a spatially integrated measure, and the cohort was restricted to persons living less than about 60 km from an airport. The effects of ozone were far from significance when only baseline means were used, but became more important than $PM_{2.5}$ for NRD mortality when continuous monthly means were used instead.

The AHSMOG authors³⁸ concluded that neither SO₂ nor SO₄²⁻ was responsible for the observed mortality associations with PM_{2.5}, which sets this paper apart from the Harvard Six Cities²¹ and ACS²³ prospective studies. Neither of those previous studies found significant effects of O₃, although all three studies used mean rather than peak O₃, which tends to have smoother spatial distributions and often masks the presence of significant daytime peaks. In contrast, the Veterans' Cohort Study³⁰ used peak O₃ and found significant associations, mainly with exposure concurrent with the period of mortality follow-up.

None of the AHSMOG findings is consistent with interpretations of previous prospective cohort studies^{21,23} that long-term exposure to air pollution creates new cases of heart disease. The AHSMOG studies are not informative about the long-term health effects of air pollution at present-day ambient levels, because of their failure to consider the much higher exposures to PM and oxidants of the 1950s and 1960s or to consider possible effects of known carcinogens such as benzo(a)pyrene (for which historical data are also available). Failure to consider a range of lag periods makes it impossible to distinguish effects of historical exposures from those of more recent times. In particular, the findings of thresholds in the responses makes it even more important to consider those prior periods during which exposure to such levels was the norm rather than the exception. Such an analysis would probably increase the implied thresholds, thus decreasing the apparent risks of present-day air quality levels. Also, CO was not considered, and the exposure lag period may have been too short to account for the latency of lung cancer. The AHSMOG studies essentially involve contrasts between northern, central, and southern California, so that spatial autocorrelation is an issue, as it is in the ACS study.^{27,28} It would have been useful to include some socioeconomic and climate-based ecological variables in the AHSMOG analyses to provide spatial context and to test for spatial confounding.

A New Cross-Sectional Analysis of Canadian Mortality Data. The Canadian Institute of Health Information released statistics on mortality rates, life expectancy, smoking habits, and other lifestyle parameters by Province.³⁹ Data on PM_{2.5} and SO₄²⁻ air quality from 1984-95 were available separately.⁴⁰ Simple regression analysis was used (for the purpose of this critique) to determine whether mortality relationships existed similar to those shown in the Harvard Six Cities²¹ and American Cancer Society²³ studies. The health endpoints investigated were age-standardized circulatory deaths, age-standardized lung cancer deaths, and life expectancy after age 65. Because data were only available for 8 provinces, the analysis was done with smoking and air quality as joint predictors, and as a 2-stage process with adjustment for smoking as the first stage. While smoking was a significant predictor for all 3-health endpoints in this very sparse dataset, air quality was not (nor was it even close).

Conclusions from Long-Term Mortality Studies. None of the additional long-term mortality studies supports the previous findings^{21,23} of associations with sulfates. The findings for PM_{2.5} in these additional studies are problematic at best.

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Conclusions

This critique has shown the following with respect to the CATF/Harvard claims and its supporting information:

1. There is no specific experimental evidence that the operation of the extant coal-fired power plants in the United States has caused violations of current ambient air quality standards or contributed to air quality deterioration. Ambient air quality (and public health) has improved and continues to improve during the period of operation of these facilities.
2. The methods used to predict air quality levels/concentrations from these plants have not been validated against ground truth and are subject to substantial uncertainties. Results from two alternative models that were used differ by an average of 60%.
3. Only one of the papers and reports in question here has been published in a peer-reviewed journal. That paper based its central estimates of PM-related health effects on acute rather than chronic relationships, such that the estimated health effects were lower than the others by an order of magnitude.
4. The health effects estimates are not based on the specific compounds emitted from coal-fired power plants or on compounds that are formed downwind in the atmosphere. Instead, they are based on the premise that all constituents of PM₁₀ are equally toxic, a premise that is without experimental support.
5. Results from six recent long-term epidemiological studies run counter to the two epidemiological studies upon which the CATF/Harvard estimates are based. Given the methodological flaws in the ACS mortality study and in its interpretation, its findings are not an appropriate basis for setting pollution control policy.
6. The most important compounds in question here are airborne sulfates. There is no support in the experimental or occupational health effects literature for the adverse health effects that are attributed to these compounds at current ambient levels.
7. Of five foreign or international bodies that have considered ambient air quality standards and health effects in detail, none has taken the position that atmospheric sulfates should be regulated.

A logical question might be, how can the peer-reviewed literature contain such conflicting information? This question was considered by Bailar,⁴⁷ who pointed out that observational studies "are subject to a great deal more variability than is captured by the usual kinds of statistical tests and confidence limits." A recent workshop⁴⁸ on the uses of epidemiology concluded that, "Determination of causation requires a weight-of-evidence approach that considers epidemiology, biologic mechanisms of action, relevant toxicology, and other factors." This critique has presented and discussed this evidence.

At the recent public discussion of EPA's revised Criteria Document for Particulate Matter,¹⁸ Dr. Morton Lippmann, a long-time advocate of the acidity hypothesis noted that "although sulfate serves well as an index of annual mortality, sulfate ion is not a causal factor but serves as a surrogate for something else." Unfortunately, this recognition has not been carried forward to its logical conclusion, which it makes little sense to regulate a surrogate agent in the absence of knowledge of the true agent for which the surrogate serves as a proxy.

The overall conclusion of the critique is thus that the CATF/Harvard claims are not supported by the totality of the science.

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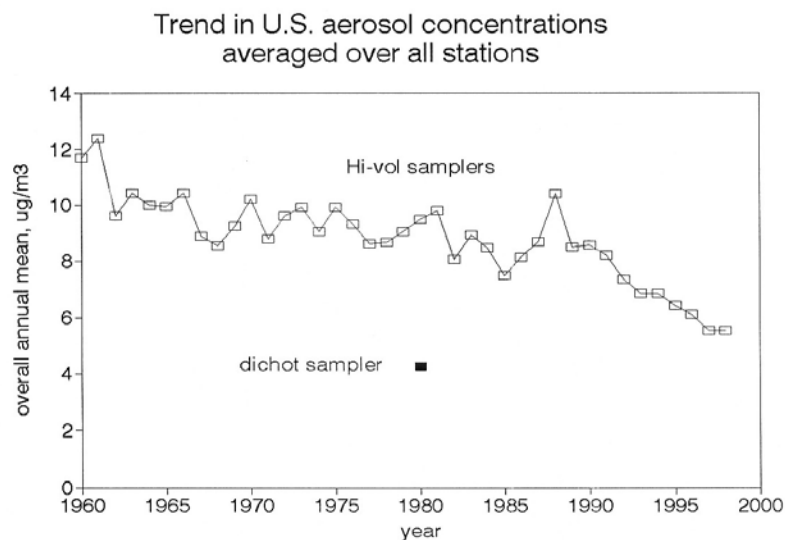
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Figure caption

Figure 1 Trends in measured ambient sulfate concentrations (data from the EPA AIRS database).



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Appendix A - Summary of the Evidence for Health Effects of Sulfur Oxides

Studies of environmental health effects may be separated into two groups, by certainty of exposure. In clinical (human volunteers), animal toxicology, and occupational studies there is some assurance that the subjects have actually been exposed to the agent in question, and generally without interference from exposures to other pollutants. This is certainly not the case with epidemiology studies under ambient conditions, in which many different components of the urban air pollution mix may be involved. This section of the critique deals with both categories of studies.

Background

While SO_2 has long been known to be toxic at sufficiently high concentrations, the main emphasis with SO_x has been on the oxidation products, collectively known as sulfates and measured in the ambient in terms of ionic concentrations (SO_4^{2-}). Some analyses of the infamous London fog episode implicated sulfuric acid,⁴⁹ but most analysts focused on "black smoke" (carbonaceous PM).^{14,50} Early cross-sectional epidemiological studies of U.S. mortality implicated SO_4^{2-} , as extracted from TSP filters.⁵¹ However, those studies were later discredited because of their neglect of many confounding factors such as regional differences in smoking habits and inappropriate handling of the sulfate data in the statistical analysis.^{14,52} The acidic properties of some of these compounds, notably sulfuric acid (H_2SO_4) and ammonium bisulfate (NH_4HSO_4) received a great deal of attention in conjunction with the acid rain program of the late 1980s, such that the issue was reframed as one of "acidic aerosols" (H^+) rather than as "sulfates" (SO_4^{2-}).⁵³ Clinical research showed that H_2SO_4 levels had to exceed about 100 ug/m^3 in order to elicit meaningful respiratory responses;⁵³ subsequent exposure studies showed that such levels are about an order of magnitude higher than outdoor levels in the U.S. and probably two orders of magnitude higher than indoor concentrations. Further, the endogenous ammonia present in humans effectively neutralizes most of the acid aerosol that could be inhaled. It therefore should have been no surprise that recent epidemiology has failed to implicate H^+ as an important PM component that might affect human health.⁵⁴

Epidemiology

Statistical Methods of Comparison. Although each independent variable must attain a certain statistical significance level in order to be taken seriously, comparison of the attained significance levels is not a valid measure of importance relative to one another, since distribution characteristics also affect significance. Measures of the relative magnitudes of the responses to the variables, such as the attributable risk or the response associated with the median or interquartile range, should be considered as well. Sample size (degrees of freedom, statistical power) must be taken into account when comparing data sets or subsets. Although uncertainty is inevitable in epidemiology, it is important to distinguish bias from random error. For example, it may be safely assumed that results from single-pollutant regressions will be biased high. Joint regressions of pollutants with different degrees of measurement error will tend to favor the one with less error, and this concept also applies to non-pollutant confounding variables such as socioeconomic status in cross-sectional regressions.

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A previous paper²⁴ examined 50 epidemiological studies involving either SO_4^{2-} or H^+ or both; in about half of them, the authors concluded that either or both of these species were causally linked to the health effects in question. Many of these conclusions were based on single-pollutant regressions and most of them involved statistical significance criteria alone. The extents to which the inherent episodicity or differences in measurement errors of the predictors contributed to these significance levels were not considered. Attributable risks (ARs) were considered in only a few instances.

Bates⁵⁵ and others¹⁸ have argued that coherence among various health endpoints is a criterion for a causal interpretation in epidemiology. However, to be convincing, coherence must be quantitative; the magnitudes of the effects must be consistent with their relative severity. Also, the findings must be robust to alternative models and to inclusion of co-pollutants.

Results from Epidemiology Studies. Typically, various air pollutants in the U.S. have been associated with up to 4-5% of daily mortality^{14,54} (attributable risks); the implied chronic responses range from about this range³⁰ to 5 times as much²¹. In time-series studies,^{56,57,58} the attributable risks of sulfate are typically only 1-2% of daily mortality, suggesting that this results from the correlation between sulfate and other components of the urban air pollution mix. Further, since the relevant SO_4^{2-} compounds are water-soluble (by virtue of the analytical methods used), acute responses due to transient irritation should logically be more severe than any sort of responses resulting from long-term build-up, especially given the fact that the sulfate ion occurs naturally in body fluids.^{59,60}

The ARs for hospitalization tend to be similar to those reported for acute mortality.¹⁴ However, because hospitalization is a less severe endpoint than death, it would be expected that hospitalization responses to substantially exceed mortality responses, and one would conclude that quantitative coherence is questionable.

For respiratory endpoints, the putative chronic effects are about four times the size of the acute effects, which was also the case with chronic vs. acute mortality, which is counterintuitive since one would expect greater population sensitivity to acute air pollution incidents. For mortality, cohort²⁸ and ecological²⁵ studies have demonstrated the existence of regional spatial confounding, which suggests that such confounding may exist for long-term cross-sectional studies of lung function and respiratory symptoms as well.

Experimental (Toxicology) Studies

Most of the toxicological studies of sulfate compounds have involved acidic species, with $(\text{NH}_4)_2\text{SO}_4$ serving as a control species. In one of the more recent such studies, Schlesinger and Chen⁶¹ found that H_2SO_4 was more potent in animal tests relative to NH_4HSO_4 than the respective H^+ concentrations would indicate. Since NH_4HSO_4 is by far the more common acidic particle species in the ambient, this suggests that toxicology and human clinical studies based only on H_2SO_4 should be interpreted cautiously in terms

of risk. Previous human clinical studies⁶² had shown that certain asthmatics were more sensitive than normals and that responses were approximately proportional to the acidity of the various sulfate compounds evaluated. However, the lowest concentration tested in these 16-minute exposures was 100 ug/m^3 , which is orders of magnitude above expected ambient levels and produced no responses from the normal subjects (2 of 17 asthmatics responded at this level).

More recently, Frampton et al.⁶³ performed similar experiments on normal and asthmatic subjects at 100 ug/m^3 H_2SO_4 in conjunction with ozone from 0.08 to 0.18 ppm. Exposures were for 3 hours and included intermittent exercise. Again, there were no effects on normal subjects, but asthmatics responded more strongly to ozone in the presence of H_2SO_4 . The authors reported "no direct response to aerosol exposure" for normals or asthmatics.

Some ongoing toxicological research efforts are based on ambient particle concentrators, in which sequential virtual impaction is used together with partial withdrawal of the carrier gas (ambient air).⁶⁴ Three such stages increase the ambient particle concentrations by about a factor of 30, while maintaining particle mixtures that are representative of the local ambient air. Such a device has been used to acutely expose animals that have been compromised to represent human subjects with pre-existing cardio respiratory disease, and responses have been obtained that included acute mortality. However, the responses do not seem to correlate with the total mass of fine particles nor with their sulfate content.⁶⁵ A subsequent paper on this work⁶⁶ found no significant differences between concentrated and sham exposures taken as a group, but that there were some differences according to the constituents of the concentrated exposures. Effects of sulfur were seen only when combined with traffic constituents (Br, Pb, C) in a factor analysis.

Longer-term exposures to sulfates and acids have been evaluated in animals. Rats exposed to two levels of ammonium sulfate (70 and 20 ug/m^3) for 4 hours per day for 32 days showed no adverse effects at the low level, with or without accompanying high levels of ozone.⁶⁷ Heyder et al.⁶⁸ exposed beagle dogs to a combination of neutral sulfate particles and particulate H^+ ($15,000 \text{ nmol/m}^3$) for 13 months and found that the adverse respiratory effects seen in a previous study with sulfite alone⁶⁹ were either less detectable or reversed when H^+ was added. They concluded that it is "very unlikely that respiratory diseases can be initiated by the inhalation of these particles." An earlier canine study⁷⁰ showed no morphological changes after 620 days at 900 ug/m^3 H_2SO_4 , 5.1 ppm SO_2 , and $13,100 \text{ ug/m}^3$ of PM.

The importance of the caution has been shown in several studies of sulfate toxicity. Mice were found to be more susceptible to infection after exposure to zinc sulfate or zinc ammonium sulfate, but not to ammonium sulfate.⁷¹ Veal calves sickened and died when excess zinc sulfate was added to their feed.⁷² Residents near a dry lake from which alkaline sulfates were eroded by wind (RSP concentrations as high as 800 ug/m^3) showed respiratory symptoms such as coughing and wheezing, but no effects on pulmonary function.⁷³ Responses to zinc compounds may have been part of the reason for the high mortality seen in the Donora, PA, episode.¹⁴ Studies of the toxicity of residual oil fly ash concluded that the metal content was the critical factor, as opposed to the sulfate

content.⁷⁴ This is consistent with recent findings with respect to lung inflammation in human volunteers that were clinically exposed to extracts of PM filters containing particles associated with steel production.¹¹

Finally, it would appear that ammonium sulfate is the most relevant sulfate compound with respect to long-term exposure, both because it is more common in the atmosphere⁷⁵ and because this would be the end result of neutralization of acidic sulfates by endogenous (breath) ammonia. Ammonium sulfate is often considered "inert;" it was used as the "control" exposure in tests of the responses of elderly subjects to H₂SO₄, for example.⁷⁶ Thus, on balance, there is no strong toxicological evidence implicating nonacidic sulfates as health hazards at current ambient levels.

Physiological Considerations

Sulfate is a common constituent of body fluids, including plasma, spinal fluid, and liver tissue.⁵⁹ The additional body burden from inhalation at current air quality levels is a minor fraction of these body stores. Further, sulfate is a common constituent of medications; MgSO₄ is routinely administered intravenously as cardiac therapy,⁷⁷ as an inhalant in asthma therapy,⁷⁸ and as a neuroprotective agent.⁷⁹ Magnesium sulfate excretion has been reported to be prompt;⁸⁰ around 70% of doses were excreted within 72 hours, which would imply that any putative long-term effects of sulfate intake would not be manifested by metabolic build-up. It is thus difficult to conceive a mechanism for long-term responses to (soluble) sulfate inhalation at typical ambient levels.

Occupational Studies

Occupational exposure studies may also be useful in this regard. A study of sodium sulfate miners⁸¹ showed no adverse effects in terms of lung function, hypertension, blood chemistry, or urinary sulfate content, both in terms of consistency with normal ranges and in terms of differences according exposure duration. Sulfate dust exposures of such occupations are commonly in mg/m³ levels. Exposures to alkaline sulfates in the paper industry were associated with lung cancer and mesothelioma, but the authors blamed concomitant exposure to asbestos, and the study did not control for smoking.⁸² A detailed review of agents associated with occupational asthma did not include any sulfate compounds or mineral acids.⁸³

The Sulfur-Transition Metal Hypothesis

One of the major unknowns inherent in the rationalization of adverse health effects associated with PM at current ambient levels is the lack of consensus on physiological mechanisms.¹⁸ Candidate causal agents include acid aerosols, bioaerosols, ultra fine particles, metals.^{18,84} Given the low probability of actual exposure of the most susceptible subjects (i.e., frail elderly) to outdoor H⁺ levels high enough to elicit responses and the poor showing of H⁺ in the epidemiological studies cited above, it would appear that acidity is also an unlikely causal factor.

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However, a potential causal mechanism for adverse health effects of PM involves acidic reactions with transition metals leading to oxidative stress and inflammation.⁸⁵ Transition metals can form complexes, sometimes involving hydrated ions. Sulfate is one of the ligands available in the atmosphere to form such complexes. Animal toxicology based on direct instillation of combustion product PM (especially residual oil fly ash [ROFA], which contains metal sulfates) has shown that such responses appear to relate to the quantities of bioavailable metals, especially iron. Since most metal particles in the atmosphere are present as oxides, solubility is an important issue and bioavailability may involve dissolution in weakly acidic body fluids. One potential environmental interaction of importance that could enhance solubility thus could be the coating of solid metallic particles with acidic sulfates such as H_2SO_4 .⁸⁶ The available data on both particulate air concentrations and concentrations in weakly acidic precipitation show that S levels far exceed those of transition metals such as Fe, Mn, V, or Zn. If the S-metal interaction involves coating a solid metal particle with either SO_2 or a liquid acidic sulfate, the relationship may be non-linear, in that excess S over and above the coating volume may have little or no effect. In this instance, reducing ambient metals would be much more effective than reducing ambient S.

However, there are many other aspects of this hypothesis remaining to be explored, including: (1) the existence of thresholds (at lower doses) due to neutralization by breath NH_3 , (2) effects of the other elements that usually accompany ambient Fe (Si, Al, Ca), evidence for H^+ coatings on ambient particles. In addition, there is no direct epidemiological support for the transition metal hypothesis, since elemental concentrations of Fe have not been shown to be better predictors of acute health responses than PM concentrations per se.⁸⁷ Of course, this could be due in part to the use of total Fe content rather than soluble Fe content as the predictor variable, or due to confounding from indoor sources of metals.

Summary

This striking lack of corroborative evidence has been ignored by CATF and other advocates who continue to associate the sulfate ion (SO_4^{2-}) with adverse health effects and to use such associations as a basis for demanding further reductions in SO_2 emissions.

What Are the Official Positions of Other Countries on the Health Effects of Sulfates?

Although the United States has been considering an ambient air quality standard for sulfates for almost 25 years,⁴² none has been promulgated, nor has any other country done so. The following position papers from other countries provide more detailed information.

The Netherlands. The (preliminary) Dutch position on PM health effects was presented in a recent draft "discussion document".⁴³ Important conclusions include that: the low acidity of Dutch ambient PM, in combination of the low pulmonary and cardiovascular toxicological potency of acidic aerosols, leads to the conclusion that acidity is probably

not the causal factor for the PM associated health effects in the Netherlands. The main fractions of Dutch ambient particles are ammonium nitrate and sulfate, together with sodium chloride. No toxicity has been identified for these components in rodent studies after exposure an order of magnitude higher than ambient levels. Taking into account that these components are soluble in water and normal concentrations in body fluids are at least an order of magnitude higher than those that can be reached from the absorbed dose by way of inhalation, makes it highly improbable that sodium chlorides, ammonium nitrate or ammonium sulfate are the causal fractions of the PM associated health effects in the Netherlands.

Sweden. A "criteria document" for Sweden was prepared at the request of the Swedish Environmental Protection Agency and published as individual authored chapters.⁴⁴ A separate evaluation of cancer risks had been done previously. All of the important epidemiology studies that were available at that time seem to have been reviewed. The summary (p. 80) focuses on PM₁₀, but also states: "It has lately been suggested that the smaller particulate fractions (e.g., PM_{2.5} or even smaller) are primarily responsible for the health effects observed. The possibility that particles are a surrogate for other pollutants cannot be completely dismissed." Neither sulfates nor acid aerosols were mentioned in this summary.

The United Kingdom. A summary report⁴⁵ on air pollution health effects was prepared at the request of the Department of Health. The framework of the analysis included the caveat that estimates of effects would only be provided when the following were available: (1) "exposure- response relationships (coefficients) which, in the view of the sub-group, could be applied in the UK with reasonable confidence", and (2) adequate data on the distribution of concentrations of air pollutants across the country which could be combined with data on population to provide estimates of population exposure" (p.1). Estimates of national health impacts were provided for PM₁₀, SO₂, and O₃, all based on daily time-series studies. With respect to long-term (chronic) effects, the report said: "People who live in polluted areas are likely to differ in various ways from people who live in an unpolluted environment. It is not easy to allow for the confounding variables (eg, smoking, diet) particularly if they include subtle sociological and behavioral factors. This is not an issue with the acute studies, since the background factors are constant. Another problem is that information about exposure is usually only available for the present and the fairly recent past, while some of the effects may be attributed to exposure in childhood." (p. 11). This document focuses on PM₁₀ but offers the following with respect to speciation: "It is probable that the toxicity of particles varies also according to composition and surface properties, and, for example, is greater with higher acidity, and less in proportion to their solubility." (Note that all sulfates used in U.S. epidemiological studies are water-soluble, since aqueous extraction is used to remove those particles from the filters on which they were collected.) The UK report goes on to say, "There is as yet a limited amount of epidemiological evidence on the health effects of PM_{2.5}, sulphates, and other fine fractions of ambient particulate matter. The available evidence comes almost entirely from North America." (p. 14).

Canada. The Canadian counterpart⁴⁰ of an EPA PM criteria document¹⁸ noted that SO_4^{2-} comprises about 17% of $\text{PM}_{2.5}$ in Canada, while $\text{PM}_{2.5}$ is about half of PM_{10} . Although this document noted the difficulties in separating effects of PM from effects of gaseous pollutants, "Reference Levels" were recommended for PM_{10} (25 $\mu\text{g}/\text{m}^3$) and $\text{PM}_{2.5}$ (15 $\mu\text{g}/\text{m}^3$). The document noted that $\text{PM}_{2.5}$ "has been shown to have a more robust association with mortality in most studies than other fine particle metrics (such as sulphate or acidity)." (p. 18 of the Executive Summary).

World Health Organization (WHO). Information about WHO Air Quality Guidelines was obtained from their website.⁴⁶ Although guideline (ambient limits) were specified for gaseous pollutants and for Pb, no such values were given for PM. Some summary graphics of various short-term epidemiological studies were provided instead, and no guidance was given for long-term effects. No guideline information was provided for sulfates, nor were these compounds included in the section on "other" and carcinogenic pollutants.

Appendix B - Summary of the Health Effects of Atmospheric Inorganic Nitrates

The literature on possible health effects of atmospheric inorganic nitrates is sparse, for good reason. First of all, the type of nitrate compound being considered is crucial. Organic nitrates, such as nitrosamines or peroxyacetyl nitrate (PAN), which is the most abundant form of nitrate in urban air, are not directly involved with power plant emissions. The compounds of primary interest here are inorganic nitrates, chiefly ammonium nitrate that is formed from the reaction of nitric acid in the power plant plume with ammonia from ground-level sources.

Overall exposure to nitrates is primarily through ingestion. An early environmental assessment by the National Academy of Sciences estimated intake through ingestion at 100 mg/day.⁸⁸ Nitrates in drinking water have long been of concern, for example. By contrast, with a mean air concentration of 5 ug/m³, intake through inhalation would only be about 0.08 mg/day. Thus, if there were concerns about the total body burden of nitrates, it would not likely involve the inhalation pathway.

Further, some nitrogen compounds, notably NO, have been found to offer beneficial uses in medicine. The journal *Nitric Oxide* is devoted to this topic. Nitrates are also used in cardiovascular therapy,^{89,90} and have been suggested for inhalation therapy as well⁹¹ Thus, there seems to be no problem with the nitrate ion per se, and since ammonium nitrate is not acidic, it is difficult to envision a toxic role for this compound.

Epidemiology studies that investigated airborne nitrates are also relatively rare. Brunekreef et al.⁹² included sulfate and nitrates in their comprehensive study of daily mortality for the entire country of The Netherlands. Taken together, the mean effect of these two species was about 0.6%, which is about ¼ of the effect of NO₂, for example. These implied effects of SO₄²⁻ and NO₃ could easily have resulted from their correlations with other pollutants. The authors of this study felt that ozone was the most consistently associated pollutant. However, Fairley⁹³ found a somewhat stronger association between nitrates and mortality in Santa Clara County (San Jose), California, where nitrate comprises a larger fraction of PM, but where there are no coal-fired power plants. Again, it was difficult to separate the effects of correlated pollutants in this study. Finally, Linn et al.⁹⁴ tracked daily physiological changes in COPD patients and found no significant relationships with either sulfates or nitrates.

The overall conclusion from this limited investigation is that there is no medical evidence linking airborne inorganic nitrates with adverse health effects at current concentration levels.

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At BNL, Lipfert held both research and management posts involving national energy planning, with emphasis on environmental impacts. Projects were conducted for the U.S. Department of Energy, the Environmental Protection Agency, and the National Acid Precipitation Assessment Program. Prior to joining BNL in 1979, he was manager of air quality for an electric utility company, where he directed air quality impact studies and associated research efforts. Prior to that, Lipfert held research and management posts in the aerospace industry.

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