S. HRG. 107–1011

HEALTH IMPACTS OF PM–2.5 ASSOCIATED WITH POWER PLANT EMISSIONS

HEARING

BEFORE THE

COMMITTEE ON ENVIRONMENT AND PUBLIC WORKS UNITED STATES SENATE

ONE HUNDRED SEVENTH CONGRESS

SECOND SESSION

OCTOBER 2, 2002

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SECOND SESSION
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C O N T E N T S

HEALTH IMPACTS OF PM–2.5 ASSOCIATED WITH POWER PLANT EMISSIONS

WEDNESDAY, OCTOBER 2, 2002

U.S. SENATE,
COMMITTEE ON ENVIRONMENT AND PUBLIC WORKS,
WASHINGTON, DC.

The committee met, pursuant to notice, at 2:02 p.m. in room 406, Senate Dirksen Building, Hon. Jim Jeffords (chairman of the committee) presiding.

Present: Senators Jeffords and Bond.

OPENING STATEMENT OF HON. JAMES M. JEFFORDS, U.S. SENATOR FROM THE STATE OF VERMONT

Senator JEFFORDS. The hearing will come to order.

Good afternoon, everyone. Thank you all for being here today. I'm glad to have this chance to come together to learn more about the health impacts of air pollution.

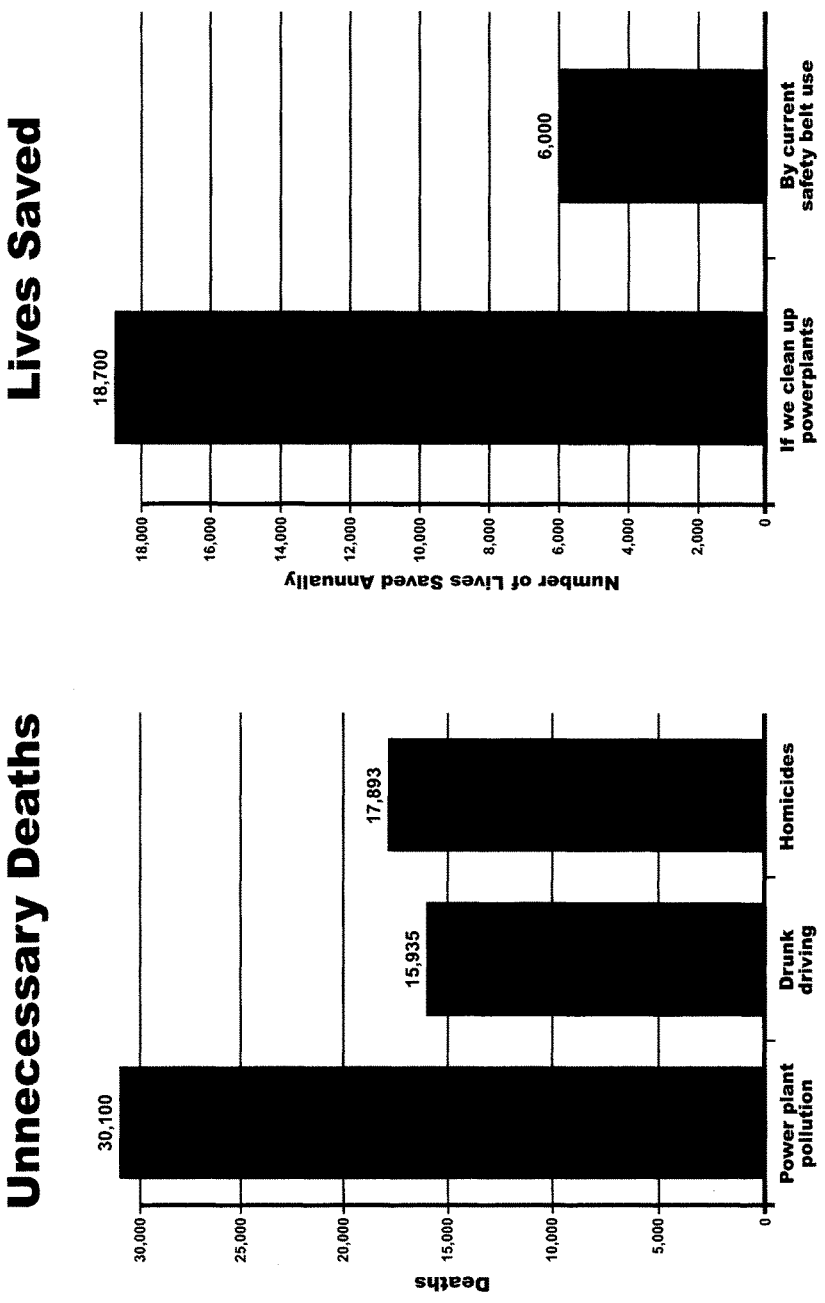
Not long ago, I was shocked to hear that as many as 50,000 people or more may be dying prematurely every year from the exposure to fine particulate matter, also known as $PM_{2.5}$ or sometimes as soot. This chart which we have up is based on the work done by many researchers, illustrating this terrible situation. More people are dying from the dirty air than are killed in auto accidents, from breast cancer and other causes. Most of this pollution comes from the burning of fossil fuel. This combustion creates tiny, almost microscopic particles from solid matter and gases. Then the wind spreads them afar and wide, sometimes thousands of miles. A few years ago, researchers documented fine particles coming from China and being deposited in the Pacific Northwest. More recently, Asian brown cloud has been in the news because of the continent-sized nature of this smog, soot and air toxics phenomenon.

Luckily, our problems are not on the scale of the Asian brown cloud any more. We can thank the Clean Air Act for that. The Act has been very effective in reducing pollution to date, and the Act provides for even greater reduction in the future if it is fully, faithfully and swiftly implemented. I hope that it will be, but the signs haven't been too promising as of late. Since the 1990 amendments, information on the health effects of fine particulate pollution have increased dramatically. Unfortunately, most of the news is bad.

In March, the Journal of the American Medical Association reported on a study which found that for increasing levels of fine particulate matter, there is a corresponding increased risk of mortality from all causes. There was an even greater risk associated with cardiopulmonary and lung cancer mortality. These findings mean

that there are practically 130 million people who live in areas polluted by fine particles who have about the same increased risk of dying from heart or lung disease as people who live with cigar or cigarette smokers and regularly experience second-hand smoke.

That's just the tip of the iceberg when it comes to the bad news. There is substantial and mounting evidence that besides death, heart disease or lung cancer, fine particles also cause decreased lung function, chronic bronchitis and aggravated asthma. Exactly how these particles cause such damage and destruction once they get deep down into the lungs is not entirely known. But what we do know with some certainty angers me. A report from the Clean Air Task Force found that fine particle pollution from power plants is responsible for as many as 30,000 deaths annually. As you can see from the chart on the left side, that's more than people who die from homicide or drunk driving accidents every year. On the right side, the chart shows how many people we could save by drastically cutting pollution from power plants. Coincidentally, those are the lives saved annually by the reductions in the Clean Air Act.


Unnecessary Deaths
Deaths
30,000
25,000
20,000
15,000
10,000
5,000
0
30,100
15,935
17,893
Power plant pollution
Drunk driving
Homicides
Lives Saved
Number of Lives Saved Annually
18,000
16,000
14,000
12,000
10,000
8,000
6,000
4,000
2,000
0
18,700
6,000
If we clean up powerplants
By current safety belt use

Most of that fine particle pollution appears to be coming from older, grandfathered power plants. Those are the ones built before 1972 that were largely exempt from applying new source performance standards. These are the same plants that are opposing the Government's efforts to make them apply new, cleaner technology when they make changes to their facilities. The Administration is now thinking of making their loophole even larger through changes in the new source review regulations. That is exactly the wrong direction. We cannot afford to increase pollution in that way. We certainly cannot afford to continue wasting the lives of people every year because of pollution that is controllable and coming from obvious sources in our own back yard.

We the Congress, the Administration, elected officials, have a responsibility to act to prevent harm to the American public when we have evidence that the threat exists. The terrible attacks of 9/11 took the lives of 2,824 innocent people in the World Trade Center. There could not be a clearer or more tangible threat to our national security. Our rapid response has reached every corner of the world and almost every facet of American life. Now it may lead us to an expanded war that could be expensive in dollars and lives.

What troubles me is that we have equal, clear evidence of the threat of death and damage occurring annually from fine particulate pollution and yet there is no huge call to action from most in Congress or the Administration. Every year New York City power plant pollution causes 2,290 lives, according to the studies we will be discussing today. Saving these lives doesn't require war, and it won't cost that much. It just requires a commitment to swift action.

Perhaps our witnesses will give us good news. Maybe the threat of the fine particulate pollution is not as bad as the headlines and the studies suggest. I hope there's a slim chance that's right, because knowingly throwing away lives when we know how to save them just doesn't make any sense.

Senator BOND.

OPENING STATEMENT OF HON. CHRISTOPHER S. BOND, U.S. SENATOR FROM THE STATE OF MISSOURI

Senator BOND. Mr. Chairman, thank you very much for calling this hearing to examine the health risks associated with fine particle air emissions. I appreciate the opportunity to come and join you with the chart presentation, because I'm going to have some charts myself. I thought we might as well keep it visual as well as audible.

My real regret is that this committee will end the session by refusing to pass three pollutant legislation that would save lives by addressing this very problem. According to EPA and information you have given, Mr. Chairman, fine particles of soot and smoke pose the greatest public health risk of any regulated air pollutant. Fine particulates are associated with tens of thousands of premature deaths per year in people with heart and lung diseases. Such emissions also lead to increased hospitalizations, emergency room and doctor visits, medication use and delays, numerous days, of missed school and work.

One major source of fine particulates is the coal-fired electric utility industry. Indeed, reports show that full implementation by

electric utilities of the Federal Government's acid rain and smog reduction program in 2007 would annually save 5,900 premature deaths and tens of thousands of respiratory illnesses associated with just 8 major coal-fired utilities. The question for us becomes why is this committee passing up the opportunity to mandate further reductions from electric utilities of the pollutants that produce particulate matter?

This year, President Bush proposed his Clear Skies Initiative to reduce emissions of nitrogen oxides, sulfur dioxides, and mercury from electric utilities. Reducing emissions of these three pollutants by over two-thirds, as the President has called for, would also produce significant fine particulate emissions reductions.

While we have made great strides in reducing air pollution since passage of the Clean Air Act in 1970, and the amendments in 1990, in which I played a role, we still have further to go. Based on the latest data, 173 counties nationwide are likely to exceed EPA's $PM_{2.5}$ fine particle health standard. The chart here behind me shows where these counties are. As you can see, 157 counties in the East and in California, well represented on this committee, and we in Missouri and Illinois in the center of the Nation have some as well.

Passage and implementation of President Bush's Clear Skies Initiative would bring 54 additional counties above and beyond what will be achieved with existing programs into compliance with the fine particle standard. This chart here shows the improvement the Clear Skies Initiative would bring to over 21 million people. You can see that only a handful of counties would remain out of compliance with the $PM_{2.5}$ health standard. These are the ozone non-attainment counties, the orange are the particulate matter 2.5 non-attainment. Red are both non-attainment counties. This is the base case for 2020. This is what the Clear Skies Initiative would do, and reduce the number of areas out of compliance with either or both by a significant number by 2020.

The mortality-related benefits from reducing fine particles under President Bush's plan are equally striking. This chart describes the number of lives saved under two different assumptions analyzing the President's plan. By 2010, Clear Skies would prevent annually between 3,800 and 6,000 premature deaths related to fine particles. By 2020, President Bush's plan would prevent annually between 7,000 and 12,000 premature deaths. Mr. Chairman, the health of my constituents in Missouri would clearly benefit under the Clear Skies initiative. Beginning in 2020, over $2 billion of annual benefits of Clear Skies would occur. Missourians would face 300 fewer premature deaths, approximately 200 fewer cases of chronic bronchitis, approximately 11,000 fewer days with asthma attacks. Missourians would suffer 300 fewer hospital days and emergency stays and emergency visits, 46,000 fewer days of work lost, 360,000 fewer total days with respiratory-related symptoms.

This is legislation that should be passed. We're not taking advantage, we're not seeking an agreement to reduce NOx, SOx and mercury. The committee is failing to take action on this legislation that would address the very health risks this hearing will examine for an unrelated reason. Some people want to hold up work on reducing the particle pollution in order to make a political point about

climate change, global warming and carbon dioxide. Count me on the health side of that equation.

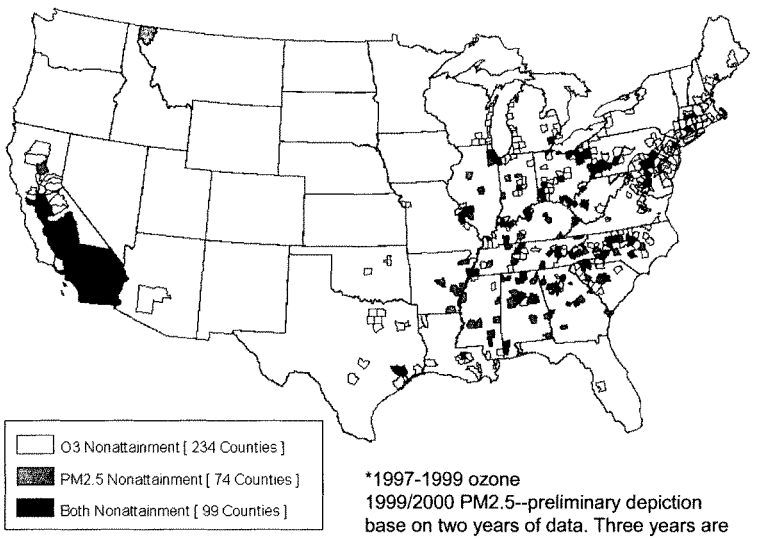
Some want to preserve the global warming issue for future elections, including the election in 2004. I urge my colleagues, as we listen to today's testimony on the health risks, to think of ways we can move forward on the three pollutant legislation. The President has put forward a plan that will save and benefit thousands. The chairman has his own plan. The opportunity exists for compromise, and I hope that we will do so next year, and I thank the chair.

Senator JEFFORDS. Thank you for your excellent statement.

Our first witness is Dr. Jonathan M. Samet, co-director of the Risk Sciences and Public Policy Institute, and professor and chair of the Department of Epidemiology, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD. Please proceed. Nice to have you here.

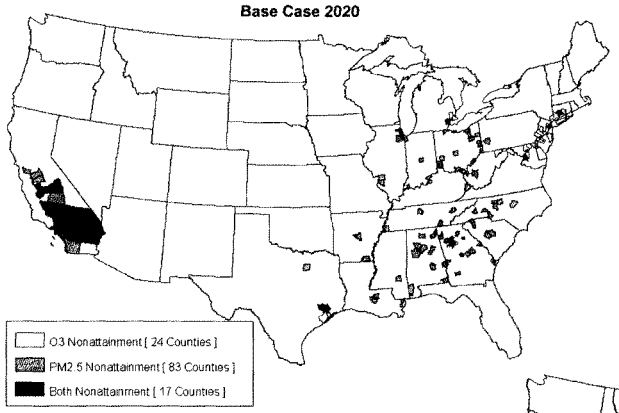
$PM_{2.5}$ and 8-hour Ozone Standards Attainment (current data*)

- Based on available 1999-2000 $PM_{2.5}$ data, 157 counties in the East and 173 counties nationwide are likely to exceed the fine particle standard
- Currently 82 million people live in 173 counties with projected concentrations greater than 15 ug/m3 (the annual fine particle standard) (59 million in the East)
- There are currently 333 counties (306 counties in the east) estimated to exceed the 8-hour ozone standard.
- Currently 120 million people live in 333 counties with projected ozone concentrations greater than 85 ppb (the 8-hour ozone standard)



*1997-1999 ozone
1999/2000 PM2.5--preliminary depiction base on two years of data. Three years are required for attainment demonstrations.

$PM_{2.5}$ and 8-hour Ozone Standards Attainment (2020)

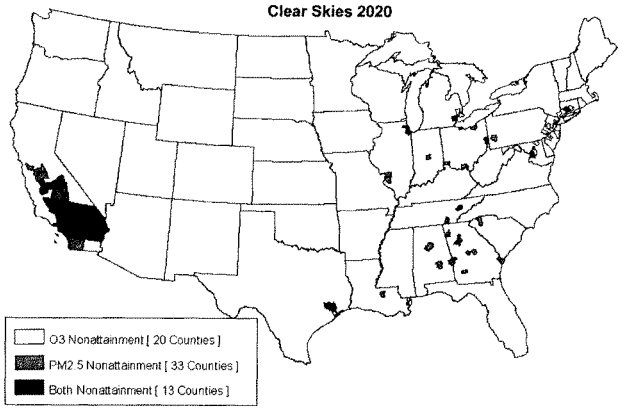


Fine particle attainment status in 2020:

- The Clear Skies Initiative would bring 54 additional counties (home to approximately 21 million people) into attainment with the fine particle standard (as compared to existing programs)

Ozone attainment status in 2020:

- The Clear Skies Initiative would bring 8 additional counties (home to over 4 million people) into attainment with the 8-hour ozone standard (as compared to existing programs)



Human Health Benefits of Reducing Fine Particulate Matter: Mortality-Related Benefits

- Clear Skies would result in significant early benefits in 2010. The total economic benefits of the Clear Skies Initiative would be $44 billion in 2010:
  - $43 billion in health benefits (as well as $1 billion in visibility benefits)
    - An alternative estimate would result in health benefits of $5 billion in 2010
  - By 2010, Clear Skies would prevent approximately 6,000 premature deaths annually
    - An alternative estimate would result in approximately 3,800 premature deaths prevented annually

- The total economic benefits of the Clear Skies Initiative would be $96 billion in 2020:
  - $93 billion in health benefits (as well as $3 billion in visibility benefits)
    - An alternative estimate would result in health benefits of $11 billion in 2020
  - By 2020, Clear Skies would prevent approximately 12,000 premature deaths annually
    - An alternative estimate would result in approximately 7,000 premature deaths prevented annually by 2020

Human Health Benefits of Reducing Fine Particulate Matter: Non-Mortality-Related Benefits

- Clear Skies would result in significant early benefits in 2010:
  - 3,900 avoided cases of chronic bronchitis
  - 5,600 fewer hospitalizations/emergency room visits for cardiovascular and respiratory conditions
  - 7.2 million fewer days with respiratory illnesses and symptoms, including work loss days, restricted activity days, and days with asthma attacks
- By 2020, Clear Skies would prevent:
  - 7,400 cases of chronic bronchitis
  - 10,500 hospitalizations/emergency room visits for cardiovascular and respiratory conditions
  - 13.5 million days with respiratory illnesses and symptoms, including work loss days, restricted activity days, and days with asthma attacks

STATEMENT OF DR. JONATHAN M. SAMET, M.D., PROFESSOR AND CHAIRMAN, DEPARTMENT OF EPIDEMIOLOGY, BLOOMBERG SCHOOL OF PUBLIC HEALTH, JOHNS HOPKINS UNIVERSITY

Dr. SAMET. Thank you, Chairman Jeffords, Senator Bond, ladies and gentlemen. Thank you for the opportunity to speak with you today about the health effects of particulate matter, and particularly fine particulate matter in the air arising from power plant emissions.

Just briefly as background, my training includes specialization in internal medicine and pulmonary diseases, quite relevant to the topic we're discussing, as well as epidemiology. I've been involved in the studies of the health effects of air pollution for more than 20 years, initially doing work in Steubenville, OH, and then in western Pennsylvania, where we carried out a series of studies to assess the effects of large coal-fired power plants on the respiratory health of women and children in the surrounding communities.

More recently, I've been involved in a project funded by the Health Effects Institute known as the National Morbidity, Mortality and Air Pollution Study. We've been using publicly available data to try and provide a national picture of the health effects of air pollution.

I've served as a consultant member of the Clean Air Scientific Advisory Committee, or CASAC, of the EPA on the particulate matter issue. Presently, I chair the National Research Council's Committee on Research Priorities for Airborne Particulate Matter. I'll be speaking, as does my testimony, to the question of, is there a hazard from fine particles? I know that others who will follow will talk about the actual magnitude of the hazard posed by particulate matter in the air.

This is a substantial research challenge, but one that the scientific community has now been addressing for decades. Part of the challenge is that the particles in air exist in a complex mixture that includes other pollutants, like ozone, and they themselves are a mixture coming from different sources. Particles are described by their size. This is important, because size is a determinant of how long particles will remain suspended in the air, whether they will reach the lung, and where they will deposit once inside the lung.

These characteristics are relevant to health, and appropriately the Environmental Protection Agency has now focused in on measuring and setting a standard for the finest particles, $PM_{2.5}$, which are in a size range that can reach into the lung. I would point out that while $PM_{2.5}$ measurements, as part of our national monitoring, are recent, PM_{10}, which has been monitored nationally since 1987 and which is the basis for much of our research, includes these smaller particles, so that studies of PM_{10} are relevant to $PM_{2.5}$.

I would also point out that we breathe particles wherever we are, including this room today, I'm sure. But the particles we breathe indoors have not only indoor sources, but they include particles that have outdoor sources. Particles and other air pollutants are important potentially to our health, because even though concentrations may sometimes be portrayed as low, we breathe in large quantities of air, 10,000 liters of air a day, approximately. We call on our lungs to handle all those particles. At times, because

of the numbers of particles or because of the toxicity of those particles, injury may result. We think that key to the action of particles is some form of inducement or stimulation of inflammation within the lung.

We know that the deposition of particles within the lung can cause adverse health effects. We can look back 50 years to the air pollution disasters in Donora, PA and in London. There was no need for complicated statistical methods to count those who died from the air pollution. The excess numbers were substantial.

Now, as levels have dropped, fortunately, researchers have faced a challenge to continue to track the health effects of air pollution and to sort out the effects of the different pollutants present in air. For this purpose, we use many different kinds of science. We use epidemiological studies like those that I carry out. We look to toxicologic evidence that tells us how particles or other pollutants cause their injury. We put all of this evidence together, so that our decisions about the health effects of particles or other pollutants do not rest on any particular study or any particular line of evidence, but on the whole body of evidence.

That body of evidence is now large, with literally thousands of studies published on particles. My written testimony includes some general references that provide access to those studies. Of course, they are cataloged by the Environmental Protection Agency in its now massive Criteria Document most recently on particles. But just to summarize a few of the things that we know at present, the numbers of deaths per day. Here we know that if we look to the numbers of deaths per day and examine the association or correlation with air pollution on the same or recent days, something that we've done in our NMMAPS project, we do find an association. Although we've recently needed to revise our estimates downward because of technical issues, we find nationally that an increase in PM_{10} is associated with an increment in the number of total deaths and an even steeper increment in the number of deaths for cardiovascular and respiratory or lung conditions each day.

You already mentioned the recent report in March in the Journal of the American Medical Association. This is one of several studies showing that in fact, for the longer term, air pollution, particulate air pollution in particular, is associated with increased risk of dying. We would take the daily studies and these longer term studies as evidence that particulate air pollution is contributing to sufficient life shortening to be a public health concern.

There are other health indicators. Hospitalization and emergency care, again, with the numbers of visits to emergency rooms or the number of people on Medicare coming to hospitals increased as the levels of particles increased from day to day. We have further evidence that people with cardiovascular diseases may be adversely affected. This is one of these areas of emerging research with a number of studies going on. But the first indications from this research provide warnings.

Asthma, an all too common condition in our country, rising in children for reasons unknown, with prevalence rates of 10 percent or more. This is a susceptible group, again with evidence showing that there are adverse effects on this important and large population.

In my testimony, I've also summarized a group of epidemiological studies, some quite old now, that address the health effects of power plants directly in communities, such as those we did 20 years ago in the Chestnut Ridge region in western Pennsylvania. While these studies are quite different in their methods, my overall interpretation of this evidence is again a warning of adverse effects of coal-fired power plants on public health in surrounding communities.

So in summary, the health effects of air pollution have been a focus of research for nearly a half century. There's no doubt, there's clear evidence from the past that high levels can have direct and evident adverse effects on health. While air pollution constitutes a complex mixture with many toxic components, the evidence consistently indicates that airborne particles in our outdoor environments and urban environments have adverse effects on health associated with premature morbidity and mortality.

Based on our knowledge of how particles penetrate in the lung, these effects likely reflect the deposition of smaller particles in the size range encompassed by $PM_{2.5}$. These particles have many man-made sources, vehicles, industry and electric power generation by coal-fired power plants. Epidemiological studies of communities located adjacent to such plants show that the health of community residents can be harmed, although links to specific products of combustion cannot be readily made.

I would note that risk assessment approaches that build on these epidemiological studies have proved quite useful for the purpose of estimating the burden of disease and ill health associated with power generation in coal-fired power plants. So I will say that while research is ongoing, as it should be and as it always will, the indications at this time provide a clear warning of a threat to public health.

Thank you.

Senator JEFFORDS. Thank you.

Our next witness is Robert M. O'Keefe, vice president of Health Effects Institute, in Boston, MA. Please proceed.

STATEMENT OF ROBERT O'KEEFE, VICE PRESIDENT, HEALTH EFFECTS INSTITUTE

Mr. O'KEEFE. Thank you, Mr. Chairman. It is a pleasure to appear before you and other members of the committee to share the perspective of the Health Effects Institute on what we have learned and what we still need to learn about the health effects of particulate matter.

For the record, I am Robert O'Keefe, vice president of HEI. HEI is an independent, not-for-profit research institute funded jointly and equally by the U.S. Environmental Protection Agency and industry. We provide impartial, high quality science on the health effects of air pollution.

In 1997, the U.S. EPA promulgated a new set of National Ambient Air Quality Standards for fine particulate matter. At that time, there were nearly 40 short-term studies that found a link between daily changes in air pollution and daily increases in death and illness. There were two long-term studies, the Harvard Six Cities Study and the American Cancer Society study, which found that

those who lived in the most polluted cities had between a 17 percent and a 26 percent increase in the risk of premature death relative to those who lived in the least polluted cities.

At the same time, there were a number of outstanding questions about these studies, including the individual short-term studies that were done by diverse investigators using different methods. Would a more systematic study find the same results? Could other pollutants, which occur along with PM, be responsible for the increased mortality? And importantly, could the Harvard Six Cities study and the American Cancer Society study stand up to intensive scrutiny and analysis from new independent investigators?

Since 1997, substantial new research has been undertaken to advance our understanding. HEI alone has invested in some 40 epidemiology, exposure and toxicology studies to test the validity of these original assumptions. Key among HEI's work have been two efforts, the National Morbidity, Mortality and Air Pollution Study, or NMMAPS, which Dr. Samet alluded to, and the re-analysis of the landmark Six Cities and the American Cancer Society studies.

NMMAPS is a systematic study of air pollution, weather and mortality in the 90 largest cities across the United States conducted under HEI oversight by John Samet and his colleagues at Johns Hopkins University. It found a consistent relationship between PM and mortality in these 90 large cities, and it was not affected when other pollutants were added to the model. At the same time, this first nationwide analysis found what may be differences in levels of effects across regions of the United States that really remain to be understood.

Recently, NMMAPS investigators identified an issue with the statistical software package used by air pollution and other investigators to analyze data. In NMMAPS, this result modified the study effect estimates. With HEI peer-reviewed alternative approaches employed, they found that the mean effect estimate in the studies shifted from a .4 percent increase in mortality for every 10 micrograms of PM_{10} to a .2 percent increase. Importantly, these results remain statistically significant, and the PM effect still does not appear to be affected by other pollutants. A further report of these efforts will be provided in January.

Looking to the long-term side, in response to requests from Congress, the U.S. EPA and industry, HEI convened a detailed re-analysis of the Six City Study and the American Cancer Society studies. Given full access to all data, HEI's expert panel selected an entirely new team of investigators who assured the quality of the original data by conducting a thorough data quality audit, and tested the results against alternative analytic approaches suggested by scientists and critics alike, without substantively altering the original findings of an association between mortality and fine particles and sulfates (a form of particles created in the atmosphere primary from coal combustion).

At the same time, the re-analysis found that the effects on mortality appeared to increase for those with less education and lower socioeconomic status. Also that there was an increase, or an association between sulfur dioxide and mortality that persisted when other variables were included.

As we look ahead over the longer term, it will be important to understand whether some particles and some sources can contribute higher toxicity and should be more stringently controlled. To address questions of particle characteristics, the HEI review committee in April 2002 issued the second in its HEI perspectives series titled, "Understanding the Health Effects of Components of the Particulate Mix—Progress and Next Steps." This review, which I have provided to your staff, summarizes recent HEI and other research and lays out recommended future approaches to understand the differential effects of particles and sources.

In conclusion, we've made much progress over the last 5 years, especially in testing the validity of the short-term and the long-term epidemiology studies. We have tested a number of possible confounding factors and alternative explanations. In reviewing the latest evidence, the HEI review committee concluded "Epidemiologic evidence of PM's effects on mortality and morbidity persist, even when alternative explanations have been largely addressed."

At the same time, some new questions have arisen. In the near term, it's necessary to complete the re-assessment of NMMAPS and identify, reassess and provide peer review for other key studies. Over the longer term, important questions remain concerning the comparative toxicity of different components and sources of the PM mixture. Only through a systematic effort to test and compare the toxicity of these diverse pollutants will we be able to have the best chance of targeting future strategies to control emissions that are the most toxic.

Thank you.

Senator JEFFORDS. Thank you. Very helpful testimony.

Dr. Wyzga is the technical executive and manager of Air Quality, Health and Risk, Electric Power Research Institute, Palo Alto, CA. Please proceed.

STATEMENT OF RONALD E. WYZGA, TECHNICAL EXECUTIVE AND PROGRAM MANAGER, ELECTRIC POWER RESEARCH INSTITUTE

Mr. WYZGA. Thank you, Mr. Chairman. Thank you very much for inviting me.

Let me introduce myself a little bit by saying that I work for EPRI, which is a non-profit, tax-exempt organization that performs scientific research for the public benefit. I have worked in environmental health research for over 30 years, published over 50 peer reviewed papers on the topic of air pollution and health, and served on numerous EPA and other scientific panels. The comments that I present today reflect my personal views and judgments as a scientist.

It was suggested that I highlight some of EPRI's most important research findings on the health effects of air pollution. I will summarize these in my oral comments, but my written comments provide further detail.

There are a large number of scientific studies that report a link between air pollution and health. From this literature, I conclude that there is a clear association between air pollution and health in the United States at current pollution levels. Among the various pollutants examined, the strongest associations between air pollu-

tion and health are for particulate matter, or PM. However, as yet there is no accepted biological explanation for the link between the pollution found in the United States today and observed health responses.

Particulate matter is made up of thousands of different components from a wide variety of sources. There are limited data on the toxicity of the different components of particulate matter. Few toxicology experiments have been undertaken examining the different fractions of PM. But those that have been undertaken have found significant difference in the toxicity for different components.

The EPRI ARIES, or Aerosol Research Inhalation Epidemiology Study project was designed specifically to examine the toxicity of the various components of PM and air pollution. This study, conducted in metropolitan Atlanta, in conjunction with U.S. DOE, several universities and others, is unique in terms of the number of air quality parameters measured and the number of health effects examined. In the study, we're looking at both potential death and disease associations with air quality. For mortality of people over 65 years old, results today show a statistically significant association for several pollutants. These include $PM_{2.5}$, PM_{10}, carbon monoxide and oxygenated hydrocarbons, which are carbon containing compounds largely in gaseous form. Indeed, when we look at several analyses, the latter, the oxygenated hydrocarbons, appear to be most consistently associated with death.

The results for disease show that in general, different components of air pollution are associated with respiratory effects than with cardiovascular effects. The respiratory effects appear to be associated with PM_{10} and the gaseous pollutants, carbon monoxide, ozone, and nitrogen dioxide. On the other hand, cardiovascular effects appear to be associated with fine particles, carbon monoxide and nitrogen dioxide. However, the only fractions of $PM_{2.5}$ that show any statistically significant associations with cardiovascular effects are particles that contain organic and/or elemental carbon. There is little evidence of any health effects tied to acid aerosols, and no significant associations have been found between any health effect and total soluble metals, ultra fine particles or sulfates.

Recent concerns have been raised about some of the past applications of statistical tools to understand the air pollution-health relationship. In fact, EPA has delayed its current review of particulate matter effects until the matter is more fully understood. Our research suggests that differences and yet other statistical methods can lead to different results. It is important to understand the influence of the different statistical methods on the results of the analyses of this air pollution-health relationship.

We now have a better understanding of the relationship between average outdoor levels of pollution and personal exposure. We see, however, that there can be short periods of time when these exposures to pollution can be extremely high. We need to identify these time periods and determine whether these short periods of exposure, a very high exposure, can impact health.

There is also a great need for additional studies that focus upon the specific components of particulate matter and the relationship to human health. I would urge others, such as the EPA, to consider studies similar to ARIES in other geographic areas. We also need

laboratory studies to examine the toxic effects of specific components and the sources of PM, so that we can identify the pollution components and sources that most impact public health. We need to develop a better biological understanding of the link between pollution found in the United States today and health effects.

Finally, statistics is a wonderful tool, and has allowed us to make considerable progress in understanding the relationship between pollution and health. But it is important that we fully understand the implications and potential weaknesses associated with the tools that we use.

To recap, my main points are as follows. No. 1, air pollution likely impacts the health of individuals in the United States today. No. 2, particulate matter is a likely candidate to explain these impacts. No. 3, in our studies, when health effects are associated with fine particles, our research points strongly to particles that contain carbons as the agents of concern. In most United States cities, carbon containing particles are the largest component by weight. Gaseous pollutants may still, however, be of health concern. There is a great need to apply alternative statistical methods in analyzing data and to understand the influence of these methods. There is a strong need to identify with more certainty those specific components of air pollution that cause health effects. Finally, decreasing the nontoxic part of particulate matter will not necessarily reduce health effects.

In summary, our latest results show that when health effects of fine particles are seen, these effects are most strongly associated with specific particle constituents. This may be an important factor in designing control strategies. Further research is needed to replicate and extend these human health studies in other geographic areas. Laboratory toxicology studies are also needed to gain a better biological understanding of the observed effects.

I would like to thank the committee for the opportunity to present my views, and would be pleased to respond to questions.

Senator JEFFORDS. Thank you very much.

Mr. Rose, you're next. executive director of the Green Mountain Club in guess where—Waterbury Center, VT. Nice to have you here.

STATEMENT OF BEN ROSE, EXECUTIVE DIRECTOR, THE GREEN MOUNTAIN CLUB, INC.

Mr. ROSE. Chairman Jeffords, thank you for the opportunity to testify.

My name is Ben Rose, I'm not a scientist. The Green Mountain Club is a 93-year-old, member-supported, not-for-profit hiking club in Waterbury Center, VT, headquartered there. The mission of the club is to make the Vermont Mountains play a larger part in the life of the people by protecting and maintaining the Long Trail, which is as you know a hiking trail which runs the length of Vermont from Massachusetts to Quebec. The southern 100 miles of the Long Trail are part of the Appalachian National Scenic Trail from Georgia to Maine, and the Green Mountain Club is one of 31 local volunteer-based clubs which maintain specific sections of the AT. The Appalachian Trail is also the longest linear national park in the world.

Although most people do not associate scenic mountain ranges with smog, some of the dirtiest air in the United States is in our mountains. Mountain air contains fine particulate matter, largely sulfates derived from burning coal, as well as nitrates and ozone, by-products of power plant emissions. The air is often at its worst in the higher elevations. This is of concern to the Green Mountain Club and our sister hiking clubs as the Long Trail, the Appalachian Trail and thousands of miles of other trails beckon hikers up into the air, which we now know is of poor quality a significant amount of time.

We are also concerned at The Green Mountain Club because we hire dozens of young people each summer as ridge line caretakers, to work on the trails and to protect the unique alpine plants that exist only on some of our highest summits. These folks spend months at high elevations. They see lots of haze, and they breathe it, too.

In August 2002, during a stretch of severe haze, particulate matter and ozone smog in New England, three hikers were treated with oxygen near the summit of Mount Washington, New England's highest peak, only tens of miles from Waterbury, VT. Staff and hikers there reported nausea and shortness of breath. During the same period, vistas from New England mountaintops were shrouded in a thick, white haze. These are the same pollutants that are causing acid rain, forming sulfuric and nitric acids responsible for the high mortality rates in our high elevation spruce and fir forests.

While many studies which will be referenced by the medical researchers on this panel have linked particulate matter to asthma, heart attacks and premature death, little attention has been paid to the health effects of fine particulate matter specifically on healthy people exercising outdoors, such as hikers. The most important study to date on the subject was conducted during the summers of 1990 to 1992, when scientists from the Harvard School of Public Health and the Appalachian Mountain Club studied the lung responses of hikers climbing Mount Washington in New Hampshire to fine particulate matter and ozone pollution.

Hikers' lung functions were measured using spirometers before and after their hikes. At the same time, ozone and $PM_{2.5}$ concentrations were measured in the air at the top and bottom of the mountain. Data was also collected regarding past respiratory history and fitness levels, current smokers were excluded from the study.

In a nutshell, the results show that healthy hikers experienced measurable declines in short-term lung function related both to ozone and to $PM_{2.5}$. Although the $PM_{2.5}$ correlation did not meet the 95th percentile confidence level, the study provided credible evidence that both ozone and particulate matter independently impact hikers' lungs. It's important to note that the air quality during the study was only moderate, with 1-hour and 8-hour ozone levels and $PM_{2.5}$ well below the Federal standards. This suggests that even moderate levels of these pollutants reduced the lung function of healthy people exercising outdoors.

The study recommended "Physicians, public health officials and the general public should be made aware of the potentially serious health effects of low-level air pollutants, not just in urban and in-

dustrial regions, but specifically on those who engage in outdoor recreation in various wilderness areas."

Currently, a similar study is being conducted in the Great Smoky Mountains National Park, in cooperation with the National Park Service and Emory University. Air quality in the Great Smoky Mountains is significantly worse than the air quality observed during the Mount Washington study. The Great Smokies have experienced 140 days of unsafe air quality over the past four summers.

Senator JEFFORDS. Four summers totaling 140 days?

Mr. ROSE. Yes.

Senator JEFFORDS. Thank you.

Mr. ROSE. Old, dirty power plants are the largest source of fine particulate air pollution in the region, accounting for half or more of the fine particulate matter and most of the sulfate deposition in the Appalachians. This means that these same plants are responsible for most of the haze and the acid rain as well.

Many coal-burning plants in the region and upwind were exempted under the Clean Air Act, and have not yet installed sulfur dioxide scrubbers or NOx catalysts, even though the technology has been available for many years. Sulfur dioxide and nitrogen oxide from power plants form sulfates and nitrate particles that can be suspended in the air for weeks and transported hundreds of miles downwind into our wilderness areas, forests and parks.

Grandfathered coal plants are endangering public health, not only to those living in cities and industrial areas, but also to those of us who exercise in and enjoy the outdoors. As a hiking club, we promote the benefits of outdoor exercise and fresh mountain air. Yet we know that those who recreate in the mountains are being exposed to unhealthy air. In conclusion, current air quality and national energy policy allow unsafe levels of fine particulate matter pollution in the air of Vermont, of northern New England and of the entire Appalachian Mountain chain that is harmful to our lungs and those of our children. People throughout the Eastern United States look to the mountains for clean, fresh air. If they can't find it in Vermont, where can they go?

We respectfully ask the Senate of the United States to act in support of aggressive measures to clean up power plants as embodied in S. 556, and to reject measures that would weaken the Clean Air Act. Thank you.

Senator JEFFORDS. Thank you for your excellent testimony.

Our last witness is Dr. Jonathan Levy, assistant professor of Environmental Health and Risk Assessment, Department of Environmental Health, Harvard School of Public Health, Boston, MA.

STATEMENT OF JONATHAN LEVY, ASSISTANT PROFESSOR, ENVIRONMENTAL HEALTH AND RISK ASSESSMENT, DEPARTMENT OF ENVIRONMENTAL HEALTH, HARVARD SCHOOL OF PUBLIC HEALTH

Mr. LEVY. Thank you, Mr. Chairman, and thank you for giving me the opportunity to speak before you today. As you mentioned, I am an assistant professor of environmental health and risk assessment, and I am a member of the environmental science and engineering program, as well as the Harvard Center for Risk Analysis.

I appear before you today as a risk assessor who has evaluated the current evidence about the health impacts of power plant emissions in multiple recent analyses. My comments will focus on the implications of the health literature for risk calculations, with more detail provided in my written materials.

As a risk assessor, I believe that decisions about alternative policies for controlling power plant pollution should be based in part on a comparison of the benefits and costs of those policies, considering the magnitude and distribution of both benefits and costs. In quantifying benefits, premature mortality associate with fine particles invariably contributes a large portion of the benefits, so I focus on this literature today.

I believe that there are three crucial questions that must be considered. No. 1, Is there a threshold below which no health effects of $PM_{2.5}$ are found, and if so, where is that threshold? No. 2, Do all types of particulate matter have similar health impacts, or are some particles more toxic than others? No. 3, Which is related to one and two, would alternative control strategies have significant impacts on the magnitude or distribution of particulate matter health impacts?

On the first point, multiple recent studies have addressed this question and have found no evidence of a threshold to date. For example, the American Cancer Society cohort study found that mortality risks decreased as $PM_{2.5}$ levels decreased, down to levels below 10 micrograms per cubic meter. Similarly, multiple investigations of time series data found no evidence of thresholds for daily changes in PM levels down to extremely low concentrations. The observational evidence therefore supports the assertion that mortality risks will continue to decrease as $PM_{2.5}$ levels decreased.

The question of relative toxicity is far more difficult to answer from a quantitative perspective. When considering power plant emissions, this is essentially a question about sulfate toxicity. In the American Cancer Society and Six Cities cohort mortality studies, the two most comprehensive and representative studies to date, sulfates show a similar association with mortality as $PM_{2.5}$ with an association also seen with sulfur dioxide.

When considering the time series mortality literature, sulfate has been associated with premature mortality in the majority of studies. I would therefore conclude that while it would be anticipated that different types of particles would have different effects, there is not sufficient information to conclude that sulfates differ from average particles in either direction. It should be noted that this is not the same as concluding that all particles are identical, but rather that the best quantitative risk estimate at present is that sulfates have similar effects as $PM_{2.5}$ in general.

I address the distribution question in greater detail in my written materials, but it is worth noting that there are spatial gradients in particulate matter impacts from power plants, and that when the health literature regarding susceptible subpopulations is taken into account, these spatial variations increased. At the same time, particulate matter from power plants is transported a long distance. This makes the exposure question national rather than local in scope.

The general conclusion I would draw is that different policy structures will lead to different distributions and exposures in health risks, and that careful consideration of these distributions should be incorporated into any comparison of control strategies.

Now, what does the health literature imply for the magnitude of benefits from alternative controls? That PM contributes to premature mortality and current concentrations are above any population threshold, and any reductions in PM concentrations will provide corresponding benefits. This means that benefits can be quantified for benefit cost comparisons as done in research studies by our research team, Abt Associates, EPA and others. Combining the cohort mortality evidence cited above with atmospheric models that we have analyzed and found to be appropriate, Abt Associates estimated that power plant emissions contribute to 30,000 premature deaths each year.

The EPA has estimated that the Clear Skies Initiative would reduce this burden by about 12,000 deaths per year, with an alternative straw proposal yielding benefits of 19,000 fewer deaths per year. While these estimates are clearly uncertain, I view the calculations as reasonable central estimates that provide a crucial foundation for policy comparisons. Thus, it is reasonable to assume that the Clear Skies Initiative would provide substantial public health benefits, but that the EPA straw proposal, which is similar to the Clean Power Act, would increase those benefits on the order of 7,000 fewer premature deaths per year.

Despite the quantitative uncertainties, the qualitative conclusion that greater controls will lead to greater health benefits appears robust, implying that choices between alternative control strategies should depend on the incremental cost and benefits of increased stringencies.

In conclusion, I thank you once again for allowing me to speak here today, and I would be happy to answer any questions.

Senator JEFFORDS. Thank you again, for very, very helpful testimony. I can't thank you all enough for helping us to really get a better idea as to where we stand and what we must and should do to help make our country more habitable and safer.

I will now have some questions for you. Dr. Samet, how do scientists determine that premature mortality and heart or lung ailments are associated with air pollution and not other factors, like diet or lifestyle?

Dr. SAMET. Clearly, other factors do influence longevity and health. But in the epidemiological studies, either the daily studies, where such factors as lifestyle don't vary day to day, just implicitly takes such factors into account. In the Harvard Six Cities and the American Cancer Society studies, the longer term studies, there was an effort to take account of such lifestyle factors as smoking, obesity and some other measures and that is done by collecting information about those characteristics and then controlling for it in the analytical approach used by the investigators.

Senator JEFFORDS. Mr. O'Keefe, I know you have another engagement. So I will go to you next. What do you think are some of the remaining gaps in knowledge regarding the health effects of particulate matter pollution?

Mr. O'KEEFE. Thank you. I think one of those has been raised, and that's the important question of whether or not there is a threshold below which particle effects exist or not. As Dr. Levy pointed out, evidence presented in both time series and studies of long-term effects have not demonstrated that there is a threshold below which we see effects. That's an important area of new work to follow up on, No. 1.

No. 2, that I alluded to earlier, as we look ahead to the next generation of particulate matter research, is there an ability to tie sources of particles and types of particles with particular health impacts? This type of analysis, which won't be done soon, and is not something we need to do before taking action during the regular course of events, if current understanding leads us to that, would really allow us over the longer term, looking forward to best target control measures, to focus on sources that may be most responsive, to focus on sensitive subpopulations that might be most toxic, and perhaps to do so in a very cost-effective manner.

Senator JEFFORDS. Thank you.

Dr. Levy, in your testimony you cite several studies that distinguish the health effects of power plant emissions from natural causes like wind-blown dust. Could you please elaborate on those findings?

Mr. LEVY. There are a number of studies in which it was tried to determine which species of particulate matter have greater health effects. Some of those, like the ones that Dr. Wyzga mentioned, measure a number of different constituents, sulfates, elemental organic carbon, dust and other elements, and try to look at the effects of those. Others try to take elemental data and combine them to try to attribute them to certain sources.

So one example of the latter study was based on the Harvard Six Cities data, where they looked at a number of different elements and then combined them to look at, to attribute them to coal sources, to residual fuel oil, to automobiles, to dust and so forth. What they found is particles from motor vehicles and from coal were significantly associated with premature death, whereas crustal particles were not. That's consistent with what a number of different studies have found, really indicating that the combustion-based fine particles seem to have greater health implications than crustal particles.

Senator JEFFORDS. Thank you. Mr. Rose, have you found that visitors to the Green Mountains express concern over pollution haze and reduced vistas? Do they feel robbed of their opportunity to see what they wanted to see?

Mr. ROSE. Yes. This is anecdotal, of course, but I do talk to a lot of hikers and visitors. I hear a lot of people express disappointment at hazy vistas. I was out quite a bit this summer and I saw some days that were clear days with a lot of haze. Other people, especially people who have been coming to the Green Mountains for a long time, comment on the same thing, that generally, visibility, even on clear days, is reduced. There is a general sense that air quality in the mountains is being impacted.

Senator JEFFORDS. Thank you.

Mr. Wyzga, your testimony on the ARIES study relies heavily on draft results but does not reference published peer-reviewed articles. When will the final results of the ARIES study be published?

Mr. WYZGA. The results based on 1 year's data have been published, and they are attachment A that I submitted to my testimony. Final results on 2 years' worth of data, manuscripts are in preparation. They will be submitted to peer review publications, I'm guessing, within the next month. I am asking the investigators to get them in as soon as possible and I think it's imminent.

Senator JEFFORDS. Thank you.

Dr. Samet, you and your colleagues performed a new analysis of the NMMAPS study. What are the important conclusions of the study that remain unchanged by re-analysis?

Dr. SAMET. Qualitatively, the conclusions are unchanged. I think Bob O'Keefe already alluded to the quantitative change in our sort of national average estimate, which dropped by half when we made some changes in the statistical tools used. The same patterns were there, seemingly a higher effect of particles in the northeast region of the United States. The greater effect for deaths from cardiovascular and respiratory diseases presumably reflect in the greater susceptibility or vulnerability of people with heart and lung disease to particles than for other causes. Again, an association with particles that was robust to control for other pollutants. I think those would be the principal findings.

Senator JEFFORDS. Dr. Levy, the NAS recently issued a report concluding that EPA's mortality estimates appropriately referenced long-term cumulative studies. What are the mortality estimates from the power plant risk assessments based?

Mr. LEVY. The ones that I referenced by Abt Associates and by EPA and by our research team were based on the long-term cohort mortality studies. There are a few of those studies available. What is generally used by myself, Abt Associates and others are estimates from the American Cancer Society study in part because it's the largest, most scrutinized study to date. It also has risk estimates that are slightly lower than those found from the Harvard Six Cities study, so it reflects a somewhat conservative interpretation of the literature.

Senator JEFFORDS. Mr. Rose, with environmental effects aside for the moment, could you tell us a little more about how severe pollution days affect the ability of volunteers to maintain trails, and how pollution might affect your business, tourism and the local economy as well?

Mr. ROSE. Well, again I would say, "yes, that we can foresee a day when many of our volunteers won't go out." In point of fact, in the last few years we've seen hiker days flat or declining in many parts of the State. We speculate as to why that's happening. Part of it is weather related, and it fluctuates from year to year. Part of it is because it's been so hot in southern New England that people are probably home in front of their air conditioners.

We actually saw a big slug of hikers come out over Labor Day weekend this year, I believe because people had stayed home all summer and said, "Wow, the summer got away from us, it was really hot." What's true for hikers is true for volunteers.

I should note that the average age of Green Mountain Club members and logically, of the volunteers who are a subset of those members, is 52. It makes sense that at that point in people's lives they have some time to give back to the trail, and are able to participate as volunteers. We see a lot of our best trail maintainers are people in their 60's and 70's. We have a lot of people who are models of good, healthy aging in the Green Mountain Club and in other hiking clubs. Those folks breathe hard when they're going up the trail.

So when we see that air quality is having an impact on people when they hike, the same is certainly true for volunteers, and it would have an impact on the long-term health of the trails that give people access, sure.

Senator JEFFORDS. Mr. O'Keefe, you mentioned a number of factors causing particulate matter toxicity. Would you tell us, in your opinion, which of these is being addressed rigorously by current regulations and which need further regulatory attention or research?

Mr. O'KEEFE. Well, you really raised the key question that the scientific community is working very hard to answer. The current National Ambient Air Quality Standards are mass based standards. By taking that approach, they act to reduce particles more broadly across the large spectrum with PM_{10}, $PM_{2.5}$ and smaller.

Within that, there are numbers of questions about which type of particle within that range could be most toxic or not. There are carbon particles, there are sulfate particles. There are biogenic particles, there are different metals that travel with particles. This area is very much an active area of research. I alluded to an understanding of the active agents in particulate health effects. They could help protect public health in the most cost-effective manner.

I will add that a mass-based standard, although it doesn't necessarily fire the bullet with ultimate precision, does have measurable effects in reducing health impacts.

Senator JEFFORDS. Mr. Wyzga, despite the clear linkage between particulate pollution from utilities and adverse health effects and death, you are recommending that this committee consider the culpability of power plant emissions. Would all the ARIES study researchers agree with your policy?

Mr. WYZGA. I really can't speak for the researchers. I think that what we're finding in the area that is important is we're finding that there are health effects at contemporary levels of pollution in the United States. I think that's something that's being widely found. We're seeing that the gases are important, as well as particles. That means we can't ignore the gases. We're seeing that different particles have different toxicities. I think it's important to really basically replicate this study in lots of other areas and see whether or not we find similar results.

I think when we look at that, we're going to be able to target specifically those pollutants that are causing our health problems. I think it's clear, and I would agree with what others have said, if we look at the data, there doesn't appear to be any threshold. It looks as if we're seeing health effects down to background and zero levels of pollution. People are dying. It's a potentially very serious—looks like a serious public health problem. To get the results,

we've got a lot more work to do in terms of targeting those specific sources and pollutants that are going to give us the biggest bang for the buck. I would urge that everybody work together to resolve this issue.

Senator JEFFORDS. Thank you. Dr. Samet, isn't it true that long-term studies examining the combined effects of chronic and acute exposure would generally yield estimates on an order of magnitude higher than the short-term studies, such as NMMAPS?

Dr. SAMET. I think one of the difficult areas where we have a signal from the long-term studies that, in terms of the effect of particles on mortality, it's about 10 times that we see in the daily time series. I would again—both estimates are in the wrong direction, that is, they're signaling an effect of air pollution on mortality, either short term or long term. We haven't quite been able to rationalize why we're seeing a seemingly larger effect on the longer term than on the shorter term. Actually, I think there will be further research, there's certainly further research on this. This is an area of research need, but it's hard to harmonize these two pieces of evidence at present.

Senator JEFFORDS. Dr. Levy, you completed new research this year that for the first time ever shows the disproportionate health impacts from power plant pollution on poor minority populations. Would you elaborate on your findings?

Mr. LEVY. Sure. This is a study that we did based on the Washington, DC. area, looking at five power plants in and around, in about a 50-mile radius around Washington, DC. What we did is look to the health literature, to the existing epidemiological studies that focused on susceptible subpopulations. So as was alluded to earlier, the American Cancer Society cohort study looked at the effect of educational attainment on the risk of mortality from air pollution, and found that those with less than high school education were much more affected than those with higher education. Similarly, it's well known that asthma prevalence, for example, and asthma emergency room visit rates are much higher in African American populations.

So they took that as a foundation for our analysis to quantify the magnitude and distribution of health benefits that would accrue if emission controls were placed on these five power plants, and found essentially that when you take the susceptibility into account—what's been documented in the health literature—that the picture changes somewhat. So if you look at the example of mortality, the method that is usually used is to assume that everyone implicitly is equally at risk. The reason, we looked at 25 percent of people had less than high school education, so normally you would assume that, well, 25 percent of benefits would accrue in that population. In fact, when we took account of the information about susceptibility, more than half of the benefits accrued in that group. You can tell a similar story for cardiovascular hospital admissions among diabetics and asthma emergency room visits as a function of race.

So we were building on the epidemiology, so clearly, more epidemiology, more studies of this type are needed to be able to provide a more robust picture. But we think this is an important direction to consider to better target who are the susceptible subpopulations,

that are their characteristics. That can potentially help us guide our control strategies.

Senator JEFFORDS. This is a question for all of you. As you know, I have been deeply concerned to learn about the health studies that show tens of thousands of lives are ended prematurely each year due to air pollution, especially from power plants. Do any of you know of other peer reviewed studies that would dispute these findings? We'll start with Dr. Samet.

Dr. SAMET. Not really, no. I think there is substantial literature, that I think have just voiced a consensus on what it shows now.

Mr. O'KEEFE. I would agree.

Mr. WYZGA. I think there are lots of studies out there that show relationship between air pollution at levels of experience today and health effects.

Senator JEFFORDS. Mr. Rose.

Mr. ROSE. I guess I would say that there is a large and growing literature in any literature where there's hundreds or thousands of studies, there are going to be some studies with negative findings. But I think the vast majority of studies are pointing to the direction that power plant air pollution leads to the premature mortality you described.

Senator JEFFORDS. Next question for everyone. Will reducing SOx and NOx by about 75 percent make progress in reducing the problem of particulate matter? Do you have any ideas for other ways that Congress can help minimize this public health threat?

Dr. Samet.

Dr. SAMET. I guess the first part is the easier one. Clearly, SOx and NOx contribute to the formation of secondary particles, and we think in fact these particles that have been discussed are possibly critically important to health effects. The second question, you know, what else can be controlled and how we should control it, I don't think lends itself to a quick answer. I think in fact you mentioned the gains that we've made in cleaning up the air with the Clean Air Act, and actually 1970, 1990 and prior attempts to clean the air.

I think the remainder of controls beyond what we've discussed, we'll have to take a look at what are the other contributors to particles. I think in line with what some of the other commenters said, are there particular sources that are associated with particles having particular toxicity that we should hone in on? I think the scientific community is probably not quite ready yet to say what those other sources might be.

Mr. O'KEEFE. I might answer that there are things that have been done recently in other areas. Being from a health effects institution, I won't delve too deeply into this area. But I would observe EPA's heavy duty diesel rule that was put into place and will significantly reduce particulate emissions from heavy duty diesel vehicles through the reduction in sulfur content in fuels and through innovative new technologies, which include traps and NOx absorbers.

So there do seem to be opportunities here for moving forward.

Senator JEFFORDS. Thank you.

Mr. Wyzga.

Mr. WYZGA. First of all, I think that clearly, both SOx and NOx form particulates, sulfates and nitrates. I think that one of the things that is—particularly, some of the work we've seen—NO_2 itself is a pollutant that may still have concerns. But don't forget the gas. That's one message I have.

Second, in our work, we don't see health effects per se of nitrates and sulfates. We see a stronger signal for some of the carbon containing particles. We don't really know what the sources are. One very interesting thing in Atlanta is that we see a very strong link between carbon containing particles and cardiovascular effects, emergency room admissions to the hospital.

These effects are only occurring in the winter. They're not occurring in the summer. We'd love to see what are the sources of carbon containing particles in Atlanta in the winter, and to our surprise, the No. 1 source was actually wood burning. The No. 2 source were diesel. Diesel contributes in the summer, but we're not seeing health effects in the summer.

I don't know if we're seeing this because of differences in pollution sources or differences in behavior, people may spend more time indoors in the winter in Atlanta. I don't think we have the answer yet, and I think we have to look a lot further into it.

But I think we really have to do a lot more work to sort of hone in on these things. There are a lot of studies out there. Another important source that sort of surprised me a lot in that area, in the summer months in Atlanta, whether it's causing health effects or not, is meat burning. There are a lot of fast food restaurants out there, and they don't have big chimneys. They're in our city.

Senator JEFFORDS. Mr. Rose.

Mr. ROSE. I understand the question to be, Is there anything else we can do? Of course, there's a lot that we can do in national energy policy and transportation policy. We need more stringent, in my opinion, vehicle efficiency standards. We need alternative fuels. We need renewables. People understand that it's all part of the same policy problem. The Clean Air Act exempted existing coal plants from requirements to retrofit with best available technology. Here it is, decades later, and the status quo is costing lives. I coach soccer on Sunday mornings, just like a lot of your other constituents. The parents on the sidelines agree that there's a lot of asthma, and people understand that the Clean Air Act hasn't realized its potential and that you're here fighting a much bigger game.

Senator JEFFORDS. Mr. Levy.

Mr. LEVY. I think my comments will echo what a lot of the other presenters have said. I think it's clear that these SO_2 and NOx controls from power plants, both because of the fine particle benefits, the ozone benefits, even the gaseous pollutant benefits, will clearly confer a major public health benefit. It's an important direction to head in. I think in terms of another direction, there isn't as obvious of a low hanging fruit, in my mind, but I think Bob was right to talk about heavy duty diesel on the transportation side as one of the other major contributors to combustion-related particles, to ozone, to a lot of urban air pollution. I think there's a lot of room for improvement in that direction as well.

Senator JEFFORDS. Last question. In your opinion, would the current particulate matter standards be sufficiently able to meet the

Clean Air Act mandate of protecting sensitive populations with an adequate margin of safety? If not, do you think EPA should consider a stricter standard?

Mr. O'KEEFE. I have to leave now.

[Laughter.]

Dr. SAMET. I have to leave before him.

[Laughter.]

Dr. SAMET. I'll just comment. This language is very difficult to interpret. In fact, I have chaired a committee of the American Thoracic Society, which wrote a statement on what constitutes an adverse health effect of air pollution, where we grappled with some of the complexity of the language of the Clean Air Act, in part around the issue that you raised. I think in terms of achieving an adequate margin of safety, that implies that we can identify a level below which effects don't occur. We can then build in the margin of safety and say, "set a standard here."

What I think you've heard from myself and others along the table today is that we can't yet identify such a point, that the evidence, we're finding a signal of an adverse effect, even as we go down to the lower levels we have today.

So the answer right now is, we haven't identified a "safe" level of effect that would allow us to meet that margin of safety statement in the Clean Air Act.

Senator JEFFORDS. Mr. O'Keefe.

Mr. O'KEEFE. I think the threshold issue is a tough one for this particular pollutant. I know, and appreciate the nature of your question. I will say that almost as we speak, EPA, its Clean Air Act Scientific Advisory Committee and many others are sifting through the weight of the evidence that's emerged over the last 5 years to draw exactly, to make exactly this determination. That process is about two-thirds of the way through, and I will wait to hear what they say, actually.

Senator JEFFORDS. Mr. Wyzga.

Mr. WYZGA. I guess first of all, I'm going to get in trouble if I make a policy statement with my employer. So this isn't a policy statement. But I think one of the premises of the Clean Air Act is that there is some threshold below which there are no health effects. We're having difficulty basically identifying such a threshold. So I think we might have to sort of think, are there new ways to set standards.

Senator JEFFORDS. Interesting.

Mr. Rose.

Mr. ROSE. Sorry, but I don't know.

Senator JEFFORDS. Mr. Levy.

Mr. LEVY. Batting cleanup, I have to once again echo some of the other comments. I agree that what the health literature is showing, seemingly no threshold, or at least that we have not yet gotten down to a threshold, the concept of trying to then set a threshold that adequately protects sensitive subpopulations seems a bit contradictory. I agree with Ron's statement that maybe we need to start thinking of alternative ways of formulating these standards.

Senator JEFFORDS. Well, thank you all very much. This has been extremely helpful to the committee. I appreciate all the work that went into being here today. That concludes our session. Thank you.

[Whereupon, at 3:16 p.m., the committee was adjourned, to reconvene at the call of the chair.]

[Additional statements submitted for the record follow:]

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INTRODUCTION

Senator Jeffords and members of the Senate Committee on Environment and Public Works, thank you for the opportunity to speak with you today concerning the health effects of particulate matter and particularly fine particulate matter arising from power plant emissions. This topic has been a focus of my research for several decades. As background, my training includes medicine with specialization in internal medicine and subspecialization in pulmonary diseases. I also have a Masters degree in epidemiology from the Harvard School of Public Health and my career has been spent in the settings of academic medicine, largely at the University of New Mexico School of Medicine, and of academic public health, now at the Johns Hopkins Bloomberg School of Public Health where I am professor and chair of the Department of Epidemiology.

Over 20 years ago, I first carried out research directed at the health effects of particulate matter. These studies were carried out in Steubenville, Ohio, where we assessed how air pollution affected the numbers of persons needing care for respiratory and other diseases in the emergency room of the community hospital, and in western Pennsylvania, where we carried out a series of studies to assess the effects of large, coal-fired power plants on the respiratory health of women and children in the surrounding communities. With colleagues at Harvard and Marshall University, I participated in an extensive study of the respiratory health of children in Kanawha County, West Virginia, following the Bhopal episode. Since 1994, with colleagues at Johns Hopkins, my research has focused on the effect of airborne particles and other pollutants on mortality. Our most recent work, the National Morbidity, Mortality, and Air Pollution Study (NMMAPS) uses publicly available data from the 90 largest cities in the United States to provide a national picture of the effect of particles on mortality, both total and from cardiac and respiratory causes of death. I have also conducted large studies directed at indoor air pollutants, such as tobacco smoke and nitrogen dioxide.

Because of my research interest in particulate air pollution, I have served as a consultant member of the Clean Air Scientific Advisory Committee (CASAC) of the Environmental Protection Agency's Science Advisory Board for the mid-1990s review of the Particulate Matter (PM) National Ambient Air Quality Standard (NAAQS) and again for the review now in progress. I also chair the National Research Council's Committee on Research Priorities for Airborne Particulate Matter, which set out a national plan for research on particulate matter in its first report in 1998. The committee is now evaluating progress since 1998 in reducing scientific uncertainties concerning particulate matter.

WHAT IS PARTICULATE MATTER AND HOW ARE WE EXPOSED TO IT?

The air that we breathe contains myriad particles that come from numerous sources that are both natural, e.g., the abrasive action of wind, and are generated by human activity, e.g., the burning of coal in a power plant. There are both outdoor and indoor sources, such as cigarette smoking and cooking. The particles in air are a complex mixture reflecting the diversity of these sources; they vary in chemical composition, shape, and size. The particles include sand, pollen and other biological materials, carbonaceous material from combustion, and particles formed secondarily from chemical and physical transformations of gaseous emissions from combustion and other sources.

Particles are often described by their size, which is a key determinant of how long they remain suspended in the air and also of whether they will reach the lung when inhaled and where they will deposit in the lung. The size of particles is described by their aerodynamic diameter in microns, a measure that is based on equivalence to a particle having a standard size and mass. Typically, in urban air, the distribution of particles by size is trimodal. The largest size mode, generally above about 5 microns in aerodynamic diameter, primarily contains dust and other particles that have been resuspended by wind and mechanical action, e.g., motor vehicles, and also some large biological particles, such as pollens. The intermediate size mode, centered below one micron contains primarily products formed by combustion including

primary particles emitted directly by the sources, such as diesel soot, and particles formed secondarily. There may be a third size mode of very tiny particles that are the immediate consequence of combustion.

These size characteristics are quite relevant to health considerations since larger particles tend to be filtered out by defense mechanisms in the nose and upper airway, and only the smaller particles, less than approximately 3.5 microns reach the lung. The sites of deposition within the lung also depend on size; the smaller particles tend to penetrate more deeply, reaching the smallest airways and the lung's alveoli or air sacs. Thus, injury to the lungs and other organ systems from particulate air pollution is thought to result primarily from the smaller particles. There is also concern, however, that persons with asthma may be adversely affected by responses to the larger particles that reach the upper airway.

The Environmental Protection Agency has set NAAQS for progressively smaller size fractions of particles, reflecting evolving understanding of how particles affect health and also measurement capability. The first particle standard was for Total Suspended Particles (TSP), which encompassed nearly all airborne particles. That standard was replaced in 1987 by a standard for PM_{10} and the new standard for $PM_{2.5}$ was added with the 1997 NAAQS revisions. The shift towards measuring and regulating smaller size fractions is well justified by scientific knowledge of the behavior of particles in the respiratory system. The size fractions for PM are inclusive: that is $PM_{2.5}$ includes all particles below the 2.5 micron diameter cut-point and PM_{10} does include the $PM_{2.5}$ size fraction. Consequently, studies of PM_{10} can inform understanding of the health effects of $PM_{2.5}$.

We are exposed to particles in all places where we spend time, both indoors and outdoors. While we spend relatively little time outdoors, particles in outdoor air, particularly the finer particles, do penetrate indoors. Consequently, the doses of particles from outdoor sources like power plants are received not only while we are outdoors, but also while we are indoors.

HOW DO PARTICLES AFFECT HEALTH?

We inhale about 10,000 liters of air per day containing countless particles. Fortunately, the lung does have mechanisms for removing particles and for detoxifying them but these mechanisms may not be sufficient if the particles are too numerous or have high toxicity. The general mechanisms of particle toxicity appear to reflect the inflammatory responses that they evoke in the lung following deposition. There may be more specific mechanisms at play as well, reflecting immune responses to antigens or the actions of carcinogens in particles. While scientific understanding of these mechanisms is still evolving, we have evidence that particles stimulate the lung's inflammatory cells, leading to the release of various mediators that continue the inflammatory process. Particles are thought to possibly affect the heart by release of mediators into the circulation. The severity of the response to particles and perhaps the nature of the response itself are likely to vary with key characteristics of the particles, such as metal content, acidity, or the various organics that are adsorbed on the surfaces of particles. Better understanding of the toxicity—determining characteristics of particles is one of the research priorities set by the National Research Council's Committee.

WHAT DO WE KNOW ABOUT THE HEALTH EFFECTS OF PARTICULATE MATTER?

The health effects of air pollution have been investigated for about half-century, following the extraordinary air pollution disasters in Donora, Pennsylvania in 1948 and in London in 1952. These and other episodes of evident excess mortality and morbidity showed that high levels of air pollution could quickly damage the public's health. Over the 50 years that the health effects of air pollution have been investigated, we have carried out many studies in communities using epidemiological approaches to assess the health effects of air pollution, including particulate matter. One challenge faced by researchers in investigating the health effects of air pollution is to attempt to separate the effects of one pollutant from the others that coexist in the pollutant mixture that is present in the air that we breathe. Nonetheless, substantial evidence has now accumulated, much of it summarized in the references that I have cited in the bibliography for this testimony.

I will focus on summarizing the more recent literature, as the earlier studies were generally carried out at levels of air pollution that are higher than measured today and the characteristics of the air pollution mixture have changed over time, as sources have changed both in their numbers and characteristics. Because researchers often use the monitoring data collected for regulatory purposes, most of the recent evidence on PM draws on measures of PM_{10}, rather than $PM_{2.5}$ as a national monitoring network for $PM_{2.5}$ has only recently been implemented.

A 1996 review by the American Thoracic Society offered a summary of literature to that time, synthesizing the information concerning major pollutants and listing health effects among the populations at greatest risk (Table 1). The more recent scientific literature includes thousands of papers on particles, so that I can only offer a general summary of the findings. The following general conclusions can be offered based on the now available evidence:

• *Daily Mortality:* Beginning in the early 1990s, many studies carried out in the United States and other countries have linked daily mortality counts to levels of air pollutants, and specifically to particles, on the same or recent days. More recently, several multi-city studies, including our NMMAPS project, have confirmed the association of particulate air pollution with mortality from all causes and from cardiovascular and respiratory causes. As anticipated, based on the concept that persons with chronic cardiovascular and respiratory diseases are vulnerable to air pollution, the effect of particles is stronger for cardiac and respiratory causes than for total mortality. In the multi-city approach, we are able to take better control for other pollutants in assessing effects of particles than with the single city approach. In NMMAPS, we estimate that the effect of PM_{10} on mortality is an increment of about 0.2% for each 10 microgram per cubic meter increase in concentration. Chicago, for example, has about 100 deaths daily; with a 20 microgram increment in concentration, about 0.5 additional deaths are projected on average. While we and others have recently needed to update our findings because of a previously unidentified statistical issue, the findings are proving robust in showing increased daily mortality associated with air pollution.

• *Long-Term Mortality:* The daily time-series studies indicate an effect of particles on mortality rates, but the data do not provide an indication of longer-term consequences. Longer-term follow-up or cohort studies provide information relevant to the question of the extent of life-shortening resulting from particulate air pollution. The strongest evidence on this question comes from two studies: the Harvard Six Cities Study and the American Cancer Society's Cancer Prevention Study II (CPS II). Both studies show that persons living in more polluted communities tend to have higher mortality rates over the approximately two decades that the participants in these studies have been observed. These analyses take into account factors that might artificially introduce an apparent association with air pollution, such as smoking and socioeconomic status. The initial findings from the two studies were replicated with a re-analysis organized by the Environmental Protection Agency. The general pattern of findings suggests that the increased mortality observed in these studies is most strongly associated with particulate air pollution. Several other studies have now been reported and others are in progress. Gaining a better understanding of the long-term effects of particulate air pollution is another of the research priorities of the National Research Council's committee.

• *Hospitalization and Emergency Care:* Using the time-series approach, a number of investigators have addressed associations of air pollution with daily counts of hospitalizations or emergency room visits. The files of the Medicare system have been used frequently for this purpose, as they provide nearly complete coverage of persons over 65 years of age in most communities. For example, Drs. Joel Schwartz and Antonella Zanobetti at the Harvard School of Public Health and members of the NMMAPS team, analyzed Medicare data from 14 United States cities. They have found associations of PM_{10} with hospitalization for cardiovascular diseases and chronic obstructive pulmonary diseases. There have been similar findings from other investigators in the United States, Europe, and other countries.

• *Cardiovascular Disease:* Persons with chronic cardiovascular diseases, particularly coronary heart disease, have been considered as susceptible to air pollution exposure, including to particulate air pollution. A series of recent studies indicate possible adverse cardiac effects of particulate air pollution, although the evidence is still somewhat mixed and preliminary. Studies show that air pollution may adversely affect the heart's rhythm and even trigger potentially fatal rhythm disturbances in high-risk persons with implanted defibrillation devices. There are supporting experimental studies.

• *Asthma:* Persons with asthma are made susceptible to air pollution by the responsiveness of their lungs to environmental triggers. Studies that monitor the health status of persons with asthma on a day-to-day basis indicate that particulate air pollution can have adverse effects.

In summary, there is now substantial epidemiological evidence linking particulate air pollution to adverse health effects, ranging from increased mortality and life-shortening to medical morbidity in people who are susceptible because they have a chronic heart or lung disease. While few of these studies have incorporated $PM_{2.5}$ as the primary exposure indicator, our understanding of particle dosimetry in the lungs implies that particles in the respirable size range are responsible for these

effects. Emissions associated with power plants contribute to the $PM_{2.5}$ mass in many locations in the U.S.

STUDIES OF THE IMPACT OF POWER PLANTS

Studies have been carried out that directly address the health effects of coal-fired power plants on surrounding communities. In a recent review, a graduate student in the Department of Epidemiology of the Bloomberg School of Public health identified 16 publications (Table 2) describing the findings of such studies. These source-directed studies considered the effects of multiple pollutants, including particulate matter. In general, their findings indicate adverse effects of coal-fired power plants on the public health in surrounding communities.

SUMMARY AND CONCLUSIONS

The health effects of air pollution have been a focus of research for nearly a half century, giving clear evidence that the high levels of the past had obvious adverse effects on health and providing a warning that air pollution continues to adversely affect public health, even at the lower levels of outdoor air pollution today. While air pollution constitutes a complex mixture with many potentially toxic components, the evidence consistently indicates that airborne particles in urban environments have adverse effects on health, causing premature mortality and excess morbidity. Based on our knowledge of how particles penetrate into the lung, these effects likely reflect the deposition of smaller particles in the size range encompassed by $PM_{2.5}$. These particles have many man-made sources, including vehicles, industry, and electric power generation by coal-fired power plants. Epidemiological studies of communities located adjacent to such plants show that the health of community residents can be harmed, although links to specific products of combustion cannot be made. Risk assessment approaches can be used for the purpose of estimating the burden of disease and ill health associated with power generation in coal-fired power plants.

Table 1. (American Thoracic Society, 1996)

HEALTH EFFECTS OF AIR POLLUTANTS AND POPULATIONS AT GREATEST RISK*

Agent	Numbers Exposed at Levels above NAAQS and Groups at Risk†	Clinical Consequences	Comments
Ozone	69.7×10^6		
	Healthy adults and children	Decreased lung function Increased airway reactivity Lung inflammation Increased respiratory symptoms	Effects found at or below current NAAQS. Effects increased with exercise
	Athletes, outdoor workers	Decreased exercise capacity	
	Asthmatics (and others with respiratory illnesses)	(Increased hospitalizations)	Effects seen in combination with acid aerosols and particles
Nitrogen dioxide	8.9×10^5		
	Healthy adults	Increased airway reactivity	Effects occur at levels found indoors with unvented sources of combustion
	Asthmatics	Decreased lung function	
	Children	Increased respiratory symptoms (Increased respiratory infections)	
Sulfur dioxide	5.2×10^6		
	Healthy adults and COPD patients	Increased respiratory symptoms Increased respiratory mortality and increased hospital visits for respiratory disease	Highly soluble gas with little penetration to distal airways
	Asthmatics	Decreased lung function	Observations related to short-term exposures
Acid Aerosols	?		
	Healthy adults	Altered mucociliary clearance	Currently not a criteria pollutant; no NAAQS established
	Children	Increased respiratory illness	Effects seen in combination with O_3 and particles
	Asthmatics and others	Decreased lung function (Increased hospitalizations)	
Particles (PM_{10})	2.5×10^6		
	Children	Increased respiratory symptoms Increased respiratory illness Decreased lung function	Effects seen alone or in combination with SO_2
	Chronic lung/heart disease	Excess mortality	
	Asthmatics	Increased asthma exacerbations	
Carbon monoxide	19.9×10^6		
	Healthy adults	Decreased exercise capacity	Effects increased with anemia or chronic lung disease
	Patients with ischemic heart disease	Decreased exercise capacity Angina pectoris (Excess mortality)	
Lead	14.7×10^6		
	Children	Altered neurobehavioral function	Elimination of leaded gasoline has resulted in marked reduction in atmospheric levels
	Adults	Increased blood pressure	

* Effects shown at exposure levels occurring in the United States. Items in parentheses are associations that have been shown in some studies, although causation is suspected. See Table 1 for definitions of health effects. For more detailed discussion, see chapters on specific pollutants.

† Number of people residing in United States counties where exposures exceed NAAQS (9). Data are based on 1990 population data and 1991 air quality data. Does not reflect exposures indoors or to brief peak levels of pollutants outdoors.

Table 2. Attributes of Epidemiological Studies of Coal-Fired Power Plant (CPP) Emissions

Study, Location, Year Published	Design	Population(s)	Pollutants and Exposure Metrics	Health Endpoints and Metric	Confounders Addressed *	General Findings	Ref.
New Cumberland West Virginia 1972	Panel 7-month	Asthmatics residing ½ mile from local CPP; Caucasian 80% adult n=20	SO_2, TSP, nitrates, sulfates, soiling index Three monitoring locations; daily means, peaks, lags	Daily asthma or wheezing attack Diary survey with some physician diagnosis	Age, sex, smoking history, allergic history, duration of asthma, temperature, humidity, wind speed, barometric pressure	Temperature and air pollution (esp. suspended sulfates) significantly associated with attack rate	1
Chestnut Ridge (I) Western Pennsylvania 1983	Cross-sectional	Adult women residing within 20 km^2 study area containing 4 large CPP n=5,557	SO_2, TSP, NO_x, O_3, haze 17 monitoring sites; 3-hr, 24-hr, 1-yr, and 4-yr means by 36 districts; three pollution level strata: low, medium, high	Chronic cough, chronic phlegm, dyspnea grade 3, wheeze most days or nights Telephone survey using ATS-DLD-78 adult questionnaire	Age, SES, ex-cigarette smoking, cigarettes per day, years smoked, cigarette tar content, cigarette smoke inhalation, spouse smoking, length of residence in area	Small increased risk of wheeze in nonsmokers independently associated with SO_2. OR of 1.26 (all current residents) and 1.40 (residents ≥ 5 yrs) No associations identified for smokers or other covariates.	2
Chestnut Ridge (II) Western Pennsylvania 1986	Cross-sectional	Children (16-11 yrs) from public school districts within 20 km^2 study area containing 4 large CPP n=4,071	SO_2, TSP See above; exposure classified as low, medium, or high based on 3-hr, 24-hr, and 1-yr SO_2 data	Chronic cough or phlegm, persistent wheeze, chest illness within previous year preventing usual activity for ≥ 3 days, serious chest illness before age 2, pulmonary function measures (FVC, FEV, V_{max})	Age, sex, SES, maternal smoking, gas stove use, parental history of allergy or respiratory disease, person completing questionnaire	Risk of serious chest illness before age 2 associated with SO_2 strata (OR range 1.13 – 2.10, $p < 0.05$). Other endpoints not associated. Absence of chronic symptoms.	3

Study, Location, Year Published	Design	Population(s)	Pollutants and Exposure Metrics	Health Endpoints and Metric	Confounders Addressed *	General Findings	Ref.
Chestnut Ridge (III) Western Pennsylvania 1987	Panel 8-month	Sample of children from the Chestnut Ridge (II) study, from areas with consistently high pollutant levels; 3 groups evaluated (1) persistent wheeze. (2) without persistent wheeze but with cough/ phlegm for ≥ 3 months. (3) no symptoms n=128 (diaries) n=144 (PEFRs) n=122 (2nd questionnaire)	SO_2, NO_2, O_3, CoH, and Temperature Maximum hourly levels for each 24-hr period at 17 monitoring stations; minimum hourly temperature from central monitor station	Incidence rates of URI, LRI, wheeze episodes. Change in daily peak expiratory flow rate (PEFR).	Standardized for SO_2 and temperature strata, symptom occurrence on previous day, informal matching for age, sex, and geographic distribution	Air concentrations lower than expected during study period, based on previous years. No important associations relative to air pollution. Cooler temperature was associated with URI and LRI. Low participation rates.	4
Finland 6 Areas Study Helsinki region of Finland 1986	Cross-sectional	Residents (15-64 yrs) living at least 3 years in one of six "affected" communities with at least one CPP & six "unaffected" communities. n=11,310 Residents near 1 CPP followed up with medical exam (n=171).	Specific pollutants not considered. Exposure based on proximity to CPP.	Hawking, cough with and without phlegm, cough with phlegm for at least 3 months, acute dyspnea	Age, sex, smoking status.	Several relationships were identified by investigators before adjusting for smoking status. An association between prevalence of cough without phlegm and proximity to CPP for nonsmokers. No associations for smokers. Medical exam follow up produced no verification of associations other than for smoking status.	5

Study, Location, Year Published	Design	Population(s)	Pollutants and Exposure Metrics	Health Endpoints and Metric	Confounders Addressed *	General Findings	Ref.
Espoo, Finland Espoo, Finland 1984	Panel 4-month	Hospital COPD patients (n_1=43) and respiratory sensitive residents (n_2=155) living near CPP Grouped into three categories: COPD, cough, or irritative symptoms	Ambient SO_2, NO_2, suspended particles, soot; concentrations of particulates measured at 3 stations, and the others at 9 stations, recording daily, 24-hr means CPP emitted SO_2 and particulate indices based on continuous flue gas monitoring and daily coal consumption.	Daily "attacks" of cough, eye, or throat irritation Daily diaries of subjects	Temperature, geographic location relative to CPP	No association between CPP emissions and ambient air data. Temperature negatively associated with "attacks" for hospital COPD patients. After controlling for temperature, increase in risk of attacks between low vs. high days for SO2 emissions index and subjects sensitive to eye/throat irritation and for ambient soot and COPD patients (both $p<0.01$).	6
Lake Munmorah New South Wales, Australia 1993	Cross-sectional	Children (kinder-garten to year 6) from "impacted" town and reference town n_1=447 n_2=404	Measures of air pollution not evaluated.	Asthma, wheeze, bronchial hyper-reactivity, past bronchitis, asthma attacks, asthma attack severity, medication use, various allergies, dry cough at night, chest colds, eczema, episodes of abdominal pain, lung function metrics Questionnaire, skin tests, lung function tests	Age, sex, atopy, father's occupation, home smoking	Children of the impacted town were more likely to have asthma (OR= 1.97) but not more severe asthma. They were also more likely to have current wheeze (OR: 2.16) and bronchial hyper-reactivity (OR: 1.96 for both).	9

Study, Location, Year Published	Design	Population(s)	Pollutants and Exposure Metrics	Health Endpoints and Metric	Confounders Addressed *	General Findings	Ref.
Hadera (I & II) Hadera, Israel 1984	Repeated Cross-sectional	Children in grades 2, 5, and 8 in 1980 and 1983 attending school within 9 km^2 radius of large CPP n_{1980}=2655 n_{1983}=1788 Large portion of two 1980 cohorts re-examined	No specific measures provided. Three population areas designated for exposure assessment of low, moderate, high "expected" pollution	Cough, sputum, cough and sputum, pneumonia, measles, other respiratory symptoms (general), FVC, FEV_1, PFR Modified ATS-questionnaires sent home; lung function tests	School-grade, gender, SES, maternal smoking, history of family respiratory disease, respiratory disease in sibs or parents	The 1980 2nd and 5th grade cohorts were specifically examined. Higher prevalence of respiratory symptoms found in younger cohort. Mixed results among three population areas. Results suffer from exposure misclassification and a lack of exposure data.	10, 11
Hadera (III) Hadera, Israel 1984	Repeated Cross-sectional	Children in grades 2, 5, and 8 in 1980, 1983, & 1986 attending school within 9 km^2 radius of large CPP	SO_2, NO_x, O_3, CO, TSP, total hydrocarbons Twelve monitoring stations (automatic), daily concentrations Three population areas, as above.	Same as above	Same as above	Respiratory symptoms more prevalent in younger generations, with statistical significant increases for cough and sputum, and asthma for one community. Mixed results for lung function measures. Reported air quality indicated that planned gradient of pollutant levels not obtained. Associations reported to be not caused by CPP.	12
Hadera (IV) Hadera, Israel 1984	Weekly surveys for 9 years	Populations served by 8 local health clinics	Meteorological and pollutant data collected as described above. Three population	Daily health service use in clinics, total daily visits, and number of visits for respiratory tract complaints.	Seasonal trends in services, influenza epidemics	No time trends or associations observed. Possible loss of patients to other clinics and turnover of patients served make results	13

Study, Location, Year Published	Design	Population(s)	Pollutants and Exposure Metrics	Health Endpoints and Metric	Confounders Addressed *	General Findings	Ref.
			areas designated for exposure assessment of low, moderate, high "expected" pollution			difficult to interpret.	
Hadera (V) Hadera, Israel 1984	Repeated cross-sectional	5th grade cohorts of 1980, 1983, 1986, & 1989 attending school within 9 km^2 radius of large CPP	SO_2, NO_x, O_3, CO, TSP, total hydrocarbons Twelve monitoring stations (automatic), daily concentrations Three population areas, as above.	Same as Hadera I, II, and III	Same as Hadera I, II, and III	Statistically significant increases in asthma and wheezing with shortness of breath. Small, clear dose-response shown over time. Bronchitis and cough with sputum showed no important change. Ambient pollutant levels were very low during study period – increased prevalent symptoms explained by other (unmeasured) causes, according to authors.	14
Gardanne Coal-Basin near Marseille, France 1988	Panel 4-month	3rd, 4th, 5th grade school children living in various communities in a coal mining & emitting region n=450	SO_2 and respirable particulates 27 monitoring stations, 15-minute automatic	Morning cough, eye irritation, runny nose, sneezing, wheezing in chest, fever Daily diaries submitted weekly	Temperature, effect lags, copollutant	Statistically significant association detected for SO_2 and morning cough and wheezing in the chest at high pollution communities. Other positive associations reported for single communities. No lag effects found. Suffers from lack of controlling for known confounders (e.g., home smoking).	15

Study, Location, Year Published	Design	Population(s)	Pollutants and Exposure Metrics	Health Endpoints and Metric	Confounders Addressed *	General Findings	Ref.
Erfurt Erfurt, East Germany 1993	Time Series	Population of Erfurt (approx. 200,000 total), heavy coal pollution number of daily events ranged from 0 to 20	SO2 (1980-1989) and suspended particulates (1988-1989, only) One centrally located continuous monitor, daily mean and maximum 30-min concentrations	Daily mortality counts Hand counting of death certificates from local health departments	Temperature, humidity, meteorology, various lag times, short-term (2 week) harvesting, copollutant, influenza outbreaks, season	Dose response coefficients identified for SO2 and suspended particulates. SO2: 5-95% quartile increase => 10% mortality increase SP: 5-95% quartile increase => 22% mortality increase	16

* The degree to which each confounding variable was addressed varies significantly both within and among the studies. In some cases, a variable is listed that was measured and qualitatively addressed, but may not have been specifically controlled for during analysis.

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STATEMENT OF BEN ROSE, EXECUTIVE DIRECTOR, THE GREEN MOUNTAIN CLUB, INC.

Senator Jeffords, members of the committee, thank you for the opportunity to testify. My name is Ben Rose. I am the Executive Director of the Green Mountain Club, a 93-year-old member-supported not-for-profit hiking club headquartered in Waterbury Center, Vermont. The mission of the Green Mountain Club is to make the Vermont mountains play a larger part in the life of the people, by protecting and maintaining the Long Trail (a hiking trail which runs the length of Vermont from Massachusetts to Quebec) and by fostering, through education, the stewardship of Vermont's hiking trails and mountains. The southern 100 miles of the Long Trail are part of the Appalachian National Scenic Trail (AT) from Georgia to Maine, and the Green Mountain Club is one of 31 local clubs, which maintain specific sections of the AT. The Appalachian Trail is the longest linear national park in the world.

Although most people do not associate scenic mountain ranges with smog, some of the dirtiest air in the United States is in our mountains.[1] Mountain air is thick with fine particulate matter—largely sulfates derived from burning coal—as well as nitrates and ozone, byproducts of power plant nitrogen oxides emissions. Unfortunately, we know that the air is often at its worst in the higher elevations. This is of concern to the Green Mountain Club and sister organizations, as the Long Trail, Appalachian Trail and thousands of miles of other trails beckon hikers up into the poor quality air.

We also are concerned because we hire dozens of young people each summer as ridgeline caretakers, to work on the trails and to protect the unique alpine plants that exist only on our highest summits. These folks spend months at high elevations. They see lots of sulfate haze, and breathe it, too.

In August 2002, during a stretch of severe haze, particulate matter and ozone smog in New England, three hikers were treated with oxygen near the summit of Mt. Washington, New Hampshire's highest peak, only tens of miles from the border with Vermont. Staff and hikers there reported nausea and shortness of breath.[2] During the same period, vistas from New England mountaintops were shrouded in a thick white sulfur laden haze. These are the same pollutants that cause acid rain, forming sulfuric and nitric acids responsible for the high mortality rates in our high elevation spruce and fir forests.[3]

While countless studies—many referred to by the medical researchers on this panel—have linked particulate matter to asthma attacks, heart attacks and premature death, little attention has been paid to the health affects of fine particulate matter on healthy people exercising outdoors, such as hikers.[4]

The most important study to date on the subject was conducted during the summers of 1990 to 1992, when scientists from the Harvard School of Public Health and the Appalachian Mountain Club (AMC) studied the lung responses of hikers climbing Mount Washington in New Hampshire to fine particulate matter and ozone pollution.[5]

Hikers' lung functions were measured using spirometers before and after their hikes. At the same time, ozone and $PM_{2.5}$ concentrations were measured in the air at the top and bottom of the mountain. Data was also collected regarding past respiratory history and fitness levels, and current smokers were excluded.[6]

In a nutshell, the results showed that healthy hikers experienced measurable declines in short-term lung function.[7] related to ozone and as well as $PM_{2.5}$.

Note that, although the $PM_{2.5}$—correlation did not technically meet the 95th percentile confidence level, the study provides credible evidence that both ozone and particulate matter independently impact hiker's lungs. It is important to note that the air quality during the study was only moderate, with 1-hour and 8-hour ozone levels and $PM_{2.5}$ well below the Federal standards. This means that even moderate levels of these pollutants reduce the lung function of healthy people exercising outdoors.

The study recommended:

> "Physicians, public health officials and the general public should be made aware of the potentially serious health affects of low-level air pollutants, not just in urban and industrial regions but specifically on those who engage in outdoor recreation in various wilderness areas."[8]

Currently a similar study is being conducted in the Great Smoky Mountains National Park in cooperation with the National Park Service and Emory University. Air quality in the Great Smoky Mountains is significantly worse than the air quality

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[1] "Out of Sight: Haze in our National Parks: How Power Plants Cost Billions in Visitor enjoyment Clean Air Task Force for Clear the Air, September 2000. Available at: *http://www.clnatf.org/publications/reports/out—of—sight.html.* See also *American Hiker,* March/April 2002).

[2] Georgia Murray, Staff Scientist, Appalachian Mountain Club. Personal communication. September 2002.

[3] Dr. L. Bruce Hill, Senior Scientist, Clean Air Task Force. Personal communication. September 2002.

[4] "Coal blamed for haze", Atlanta Journal-Constitution. Friday, August 30, 2002

[5] "Effects of Ozone and Other Pollutants on the Pulmonary Function of Adult Hikers" by Korrick, Neas, Dockery, Gold, Allen, Hill, Kimball, Rosner, Speizer. Environmental Health Perspectives, Volume 106 Number 2, Feb. 1998. Conducted 1990–92, Pinkham Notch, New Hampshire, White Mountain National Forest by Harvard School of Public Health, Brigham and Women's Hospital and Appalachian Mountain Club. http://ehpnet1.niehs.nih.gov/docs/1998/106p93–99korrick/korrick-full.html

[6] Ibid.

[7] Ibid.

[8] Ibid.

observed during the Mount Washington study. The Great Smokies have experienced 140 days of unsafe air quality over the past four summers.[9]

Old dirty power plants are the largest source of fine particulate air pollution in the region, accounting for half or more of the fine particulate matter and most of the sulfate deposition in the Appalachians.[10] This means that these same plants are responsible for most of the haze and acid rain as well.[11]

Many coal burning plants in the region and upwind were exempted under the Clean Air Act (CAA) and have not yet installed sulfur dioxide scrubbers or NOx catalysts,[12] even though the technology has been available for many years.

Sulfur dioxide and nitrogen oxides from power plants form sulfates and nitrate particles that can be suspended in the air for weeks and can be transported hundreds of miles downwind into our wilderness areas, forests and parks.

Grandfathered coal plants are endangering public health not only to those living in cities and industrial areas but also to those of us who exercise in and enjoy the outdoors.

As a hiking club, we promote the benefits of outdoor exercise and fresh mountain air and yet we know that those who recreate in the mountains are being exposed to unhealthy air.

Current air quality and national energy policy allow unsafe levels of fine particulate matter pollution in the air of Vermont, of Northern New England, and of the entire Appalachian Mountain chain that is harmful to our lungs and those of our children. People throughout the Eastern United States look to the mountains for clean fresh air. If they can't find it in Vermont, where can they go? We respectfully ask the Senate of the United States to act in support of aggressive measures to clean up power plants as embodied in S. 556 and reject measures that would weaken the Clean Air Act.

Thank you.

STATEMENT DR. RONALD E. WYZGA, EPRI, PALO ALTO, CA

INTRODUCTION

I am Dr. Ronald E. Wyzga. I work for the Electric Power Research Institute (EPRI), in Palo Alto, California. EPRI, a voluntarily-funded 501(c) (3) non-profit organization operating in the public interest, is almost 30 years old and has an annual budget of approximately $350 million. EPRI's Environment Sector has an annual budget of approximately $50 million; this makes EPRI one of the largest privately-funded health and environmental research organizations in the world. Within the Environment Sector, I am responsible for air quality research, including research on the health effects of air pollution. The results of EPRI's health and environmental research is published and made publicly available, usually through the peer-reviewed scientific literature.

I began my research career working on the relationship between health and air pollution (specifically particulate matter) while a graduate student at the Harvard School of Public Health, and my doctoral dissertation in biostatistics in 1971 addressed this topic. Since then I have been actively engaged in environmental health issues. I have co-authored a book and published over 50 peer-reviewed papers. I have served on and chaired subcommittees of the National Research Council (NRC), National Academy of Sciences. I currently serve on the NRC Committee on Research Priorities for Airborne Particulate Matter. I have also served on or chaired several EPA Science Advisory Board Committees, and I have been appointed a Fellow of the American Statistical Association. The comments that I present today reflect my personal views and judgments as a scientist who has worked in this area for over thirty years. These comments should not be construed to be the official opinion of my employer or of any associate.

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[9] Source: Jim Renfro, Air Quality Specialist, Great Smoky Mountains National Park, National Park Service.

[10] According to the National Park Service's "Air Quality in National Parks" 2nd edition, sulfate particles formed from sulfur dioxide emissions associated with fossil fuel combustion (mostly from electric generating facilities) accounts for up to 60%–80% of visibility impairment in eastern parks compared to only 30–40% visibility impairment in western states.

[11] Abt Associates (2000). Out of Sight: The Science and Economics of Visibility Impairment, Bethesda, MD. Available at: *http://www.clnatf.org/publications.*

[12] National Park and Conservation Assoc. (NPCA) Fact Sheet.

SUMMARY

There are a large number of scientific studies that report a link between air pollution and human health. I have personally been involved in some, and EPRI has supported many more. The majority of these studies link particulate matter with health effects; however, some of these studies do not show an association with health, and other studies implicate gaseous pollutants in addition to, or in place of particulate matter. In any consideration of the health and air pollution issue, it is important to keep in mind that air pollution is a complex mixture of many different types of gases and particles. Discerning specific causative agents is a challenge we in the scientific community are working to address. Today I want to highlight some of the work that EPRI has recently been involved in to provide you with some of our latest results.

There have been several major facets to our research:

1. It is important to understand which specific components of air pollution are associated with health impacts. In studies undertaken to date, the strongest associations between air pollution and health are with particulate matter. In studies which include particulate matter (PM) and other pollutants, such as ozone and carbon monoxide (CO), in their analyses, PM is most consistently associated with health responses; there are, however, some exceptions where other pollutants, especially carbon monoxide, are most highly associated with health responses. Very few studies have considered a comprehensive set of the pollutants, especially the different chemical constituents of particulate matter, in their analyses. This is because monitoring programs currently only measure a small number of compounds.

There are limited data on the toxicity of the different components of particulate matter. Few toxicology experiments have been undertaken examining the different fractions of PM, but those that have been done have found differences in toxicity for the different fractions. Other results show that the total quantity of PM by weight does not explain biological responses. Certain components in PM appear to explain the toxicity of PM more readily than total PM.

2. The EPRI ARIES (Aerosol Research Inhalation Epidemiology Study) project was designed to examine the toxicity of the various components of PM and air pollution. This study is unique in terms of the number of air quality parameters measured and the number of health effects examined. This study, undertaken in Metropolitan Atlanta in conjunction with several universities, U.S. Department of Energy, and others, characterized the air quality on a daily or more frequent basis for over one hundred air quality variables. This characterization, accompanied by a suite of epidemiological studies, allowed us to examine the influence of the various components of air pollution on a variety of health outcomes.

In general, the ARIES study is finding that *different* components of air pollution are associated with respiratory effects than are associated with cardiovascular effects (heart-related effects). More explanation of the preliminary results is given in the detailed testimony, but in summary, the *respiratory effects* appeared to be related to the gaseous pollutants (carbon monoxide, ozone, and nitrogen dioxide) and *cardiovascular effects* appeared to be associated with $PM_{-2.5}$ (particles 2.5 microns in size and smaller) and *carbon monoxide*. However, the only fraction of $PM_{-2.5}$ that showed any association with the cardiovascular effects were particles containing organic and elemental carbon. It is the $PM_{-2.5}$ fraction that has been at the center of attention as the potential cause of negative health impacts. For *total mortality*, the pollutants most consistently associated with premature death are *oxygenated hydrocarbons*, substances that to date have had limited study.

3. EPRI has initiated smaller ARIES-like studies in Baltimore and St. Louis to determine whether the results from Atlanta can be replicated elsewhere. A major effort is also underway to launch a study very similar to ARIES in Chicago.

4. A major toxicology effort will start soon in which the effects of coal combustion emissions will be investigated by exposing animals to diluted, aged emissions from power plants. This effort will provide important data to help evaluate different combinations of fuel type, control technologies, and burning configurations. The results of this work will be particularly useful to help inform and complement the research underway at the National Environmental Respiratory Center in Albuquerque, which is also evaluating the toxicity of emissions from diesel and gasoline engines, as well as wood smoke.

5. EPRI has also been active in trying to understand the implications of alternative statistical methods used in the analyses of epidemiological data. Given the recent discovery that the applications of statistical software have led to erroneous results in some pollution health studies, the EPA is delaying its review of particulate matter health effects. Other statistical analyses require judgments that can impact their outcome. It is important to understand these potential impacts.

6. EPRI has undertaken studies to understand the nature of exposure to the various constituents of air pollution, including particulate matter and its major constituents. We have found that there appears to be a better association between personal exposure to particulate matter and outdoor measured levels than there is for many of the gaseous pollutants.

What is particularly important is that recent results suggest that there are short periods of time (in specific environments) when personal exposures to pollutants are much higher (by factors of 5 for PM and over 50 for carbonaceous particles) than the levels that we measure at our monitoring stations. We need to establish whether these short-term peak exposures are related to health responses.

7. Our joint study with Washington University of some 50,000 Veterans was designed to answer the question of whether there are long-term (chronic) effects associated with air pollution. In this study we found that after adjusting for many other factors Veterans who lived in cities with *higher levels of nitrogen dioxide* and *very high ozone levels* died earlier than those living in cleaner cities. We could find no such effect, however, when we examined particulate matter.

SCIENTIFIC ISSUES

There is a clear association between air pollution and health in the U.S. at pollution levels we have experienced in the 1990s and earlier. Several different types of epidemiological studies, undertaken at a wide variety of locations, have found associations between air pollution and human health effects in the U.S.. Among the various pollutants examined, the strongest associations between air pollution and health are for particulate matter (PM). Many of the earlier studies (pre-1990s) considered just one or a limited number of pollutants; in these studies, PM was frequently studied and found to be associated with health effects. Later studies more frequently examined multiple pollutants. Most of these studies also found associations between PM and health effects, although a subset of the studies found greater associations between health effects and other pollutants, especially carbon monoxide (CO). In interpreting the results of these studies, several factors must be taken into account. First, the pollution measurements used in these studies were made at outdoor monitoring sites; these are not necessarily representative of personal exposures to these pollutants. We now have some limited data on the differences between personal exposures and outdoor measurements. These differences are not the same for every pollutant measured, leading to possible statistical impacts on the results of the analyses of the relationships between air pollution and health.

Second, studies can only consider pollutants for which measurement data are available, and only a few pollutants/substances are generally measured. If the pollutant(s) that are truly responsible for health effects are not measured, then other pollutants that are measured and present at the same time as the responsible pollutants can be associated statistically with health effects. In such cases what we measure and use in our analyses could be a surrogate for something that is not measured. In all of our study results we need to keep this in mind. The only way to overcome this issue is to measure as many components of air pollution as possible, hopefully including the true culprit (or culprits), which only detailed analyses can reveal.

There is as yet no accepted biological explanation for the link between the levels of pollution found in the U.S. today and observed health responses. Past research has focused on epidemiological studies—observational studies on humans going about their normal activities. *Laboratory* research, which has been limited to date, can focus on establishing the underlying biological mechanisms that can cause negative health effects. Several possible biological explanations have been put forth to explain the results from epidemiological studies, and recent laboratory results support some of these hypotheses. For example, one study appeared to show that blood clotting can increase with exposure to higher levels of fine particulates. If this occurs, it could be an explanation for why some heart disease effects are related to fine particulate levels in epidemiological studies. At this time, I believe that the most likely scenario is that a combination of explanations is responsible for the effects observed, with different mechanisms acting for different air pollution/PM components. Different mechanisms may also be acting in susceptible individuals, such as asthmatics or those with hypertension. Clearly, much more work is needed to gain insight into the mechanism(s) of PM action.

Particulate matter is a complex mixture and its composition varies over time and place. Some of these major components (e.g., organic matter) contain hundreds of chemical compounds. The most important fractions of PM are carbon-containing particles and sulfate in the Eastern U.S., with carbon-containing particles being more

important in urban areas. In the Western U.S., nitrates are more important and sulfates are generally less important.

There are limited data on the toxicity of the different components of particulate matter. Several PM components have been hypothesized to play a role in toxic responses, including acid aerosols, metals, sulfates, nitrates, ultrafine particles (very tiny particles much smaller than the $PM_{-2.5}$ particles), bioaerosols (including pollen and mold spores), diesel exhaust particles, and organic compounds. Toxicological and human exposure evidence suggests that acid aerosols do not contribute much to the adverse respiratory outcomes observed in epidemiological studies; however, acid components have not been assessed thoroughly with respect to potential cardiovascular effects. Metals have been shown in multiple studies to cause cell injury and other effects. Particle size, specifically the ultrafine fraction, may also be important in the development of health effects. A number of studies have investigated the effects of ultrafine particles and have found lung inflammation and other respiratory effects, although it appears that chemical composition may play a key role in the responses observed. Cardiovascular and systemic effects of ultrafine particles have been investigated to only a limited extent. Bioaerosols are not considered to account for the reported health effects of ambient PM as their concentrations are very low and health effects can occur at times when bioaerosol concentrations are low. Toxicological evidence is accumulating to suggest that diesel PM can exacerbate the allergic response to inhaled allergenic material.

Finally, the organic compounds associated with PM have been little-studied from a toxicological perspective, although they represent a substantial portion of the mass of ambient PM (10–60% of total dry mass). Other fractions of PM, including sulfates and nitrates, appear to be of less concern.

In a recent draft report, the Netherlands Aerosol Programme concluded: "Based upon current toxicological and human clinical knowledge: water, sea salt, ammonium sulfate, ammonium nitrate, and probably non-crystalline crustal material too, can be considered an inert part of PM_{-10} at the ambient concentrations in the Netherlands." This report has not yet been finalized, and the conclusions are still under discussion.

In order to more fully understand which components of PM are responsible for the health effects observed, additional toxicological studies must be conducted. Studies which examine the toxicity of emissions from various sources of pollution can be informative in identifying those pollutants (and sources) most highly associated with health effects.

The EPRI ARIES study was designed to examine the toxicity of the various components of PM and air pollution. This study is unique in terms of the number of air quality constituents measured and the number of health effects examined. The best way to increase our understanding of the types of PM and air pollution that may be responsible for the health effects observed in other studies is to undertake a study in which all of the potentially relevant fractions of PM are measured. Traditionally we only measure what is required because of local, state or Federal regulations. On occasion a research study may measure a larger array of air pollutants, but it is rare to have a large number of constituents measured systematically over an extended period of time. ARIES addresses this need through detailed air quality characterization for a period of over two years and through undertaking several epidemiological studies to relate air quality characteristics to health effects. Appendix A provides further details about ARIES.

Extensive daily—and in some cases continuous—measurements were made for all of the particle size fractions and constituents about which concerns have been raised. At the same time, several epidemiological studies were undertaken to examine the potential health effects of the various constituents. Initial results from the analytical team focused on the subset of air pollution measures tied to the major existing hypotheses about the pollution/health relationship. Results based upon the first year's data have been published in peer-reviewed journals; two years of data have now been analyzed and manuscripts based upon analyses of two years worth of data are now under preparation for peer review. The draft results are very informative, and I would like to share them with you.

These results are complex and reflect a methodology that examined pollutants individually. Analyses which consider several pollutants simultaneously are planned and may help identify the pollution components that are of greatest concern.

• Several pollutants are statistically significantly associated with mortality of those over 65 years old; they include $PM_{-2.5}$, PM_{-10}, CO (carbon monoxide), and oxygenated hydrocarbons. When alternative statistical models were applied, the results were most consistent for oxygenated hydrocarbons, a pollutant that has not previously been considered in air pollution health studies.

Results are available for several morbidity (disease) measures including emergency room admissions to Atlanta area hospitals, unscheduled physician visits to a health maintenance organization (HMO), and responses of defibrillator devices implanted in patients with erratic heart rhythms. Preliminary analyses of heart rate variability considered only $PM_{-2.5}$ and not its components nor gases. Based on these limited data, $PM_{-2.5}$ was found to be associated with statistically-significant changes in heart rate variability.

- Lung and respiratory problems were related to PM_{-10} and to pollutant gases including ozone, nitrogen dioxide, and carbon monoxide.
- Heart disease responses were much more likely to be related to $PM_{-2.5}$, carbon monoxide, and nitrogen dioxide.
- Organic compounds were associated with several cardiovascular effects.
- When the components of $PM_{-2.5}$ were considered, the only ones found to be significant were elemental and organic carbon.
- There was little evidence of any health effects tied to acid aerosols.
- No associations were found between any health effect and total soluble metals; additional analyses are planned to look at individual metals.
- No associations were found with ultrafine particles. Since the concentrations of these particles appear to change so rapidly over time and space, it is doubtful that the ARIES study could shed much light on the effects of these particles. Nevertheless, their concentrations are unrelated to the concentrations of other particle fractions; hence it is unlikely that ultrafine particles can explain the association seen with other particles.
- No cardiovascular or respiratory effects were associated with sulfates.

ARIES did not look at sources of pollution directly. We did, however, undertake a source-attribution analysis of the organic compounds in Atlanta. Cardiovascular effects were found in the winter months only in this study. In the winter months, organic compound concentrations were tied principally to wood smoke, although diesel emissions were also a contributor. Diesel emissions were also a major contributor to organic compounds in the summer months when no cardiovascular effects were related to these compounds.

There is a great need for additional studies which focus upon the specific components of particulate matter and examine their relationship to human health. The ARIES study will provide an important piece of evidence in understanding which fractions of PM and of air pollution are the most important in affecting human health. ARIES results are from one metropolitan area, Atlanta. Atlanta is a logical place for a study; it has high pollution levels, many sources of pollution, and no unique sources of pollution that would yield a unique result. Nevertheless it is important to undertake similar studies in other metropolitan areas. We are now engaged in similar, although more limited, studies in St. Louis and Baltimore, where detailed monitoring is underway. Much of this monitoring is funded by EPA's supersites monitoring program. Undertaking such studies is expensive because the air quality monitoring itself is costly; hence, governmental resources to undertake such studies are critical.

Secondly, more *laboratory* studies are needed which examine specific fractions of particulate matter and its toxicity. Since it would be very costly and time-consuming to test all specific compounds rigorously in laboratories, special protocols should be considered which examine the mixture of pollutants associated with specific sources. For example, studies are now underway at the National Environmental Respiratory Center to examine the toxicity of emissions from several sources. EPRI is planning some similar efforts, but clearly more research is needed. There are a large variety of emissions from different sources, and we need to learn how these emissions interact with other pollution elements once they enter the environment at large.

An ongoing committee of the National Research Council, of which I am a member, will issue a report next year identifying the highest priority research needs to inform particulate matter-health policy issues.

The implications of the statistical methods used to investigate the relationship between health and air pollution need to be fully understood. A recent announcement by researchers at Johns Hopkins University raised some issues about the past use of one particular statistical approach and its related software. Fortuitously, at a meeting of EPRI researchers with our advisors, it was decided to use alternative statistical methods in our research, and we have examined these methods thoroughly. We have found that, on occasion, ARIES results, especially in the mortality analyses, can be influenced by changes in the statistical approach even when the alternative approaches are judged reasonable by statisticians. For example, carbon monoxide (CO) was found to be statistically significantly associated with deaths of those over 65 years old with one approach but not with the other. Fortunately most

results were similar across the various approaches, but because there are some differences, it is important to articulate and understand these differences.

CONCLUSIONS

1. Air pollution likely impacts the health of individuals in the U.S. today.
2. Particulate matter is a likely candidate to explain these impacts.
3. Not all fractions of particulate matter appear to be equally toxic.
4. When health effects are associated with fine particles, our research points strongly to carbon-containing particles as the agents of concern; in most U.S. cities, carbon-containing particles are also the largest particle component by weight.
5. Gaseous pollutants are still of concern and cannot be ignored.
6. There is a strong need to identify with more certainty those specific components of air pollution which cause health effects.
7. We need to understand in more detail the personal exposure of susceptible individuals to the various air pollution components. In particular, we need to identify when and where peak exposures occur and whether these peaks are important to health.
8. There is a great need to apply alternative statistical methods in analyzing data and to understand the influence of a specific method.
9. Decreasing the non-toxic part of particulate matter will not reduce health risks.

APPENDIX A

Fact Sheet EPRI

ARIES: Aerosol Research Inhalation Epidemiology Study

Atmospheric measurements, exposure assessment, and health data in Atlanta are combined to test hypotheses concerning the health effects of $PM_{2.5}$

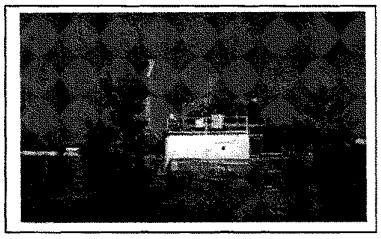

Figure 1. ARIES Core Site

Background

Findings from some epidemiology studies have shown discernible associations between daily levels of suspended particulate matter and adverse health effects. Interpretation of these associations has been difficult and controversial because particulate matter can be an index for a range of other substances in air as well as for weather and seasonal patterns.

On the basis of the epidemiology evidence, the United States Environmental Protection Agency (EPA) promulgated new National Ambient Air Quality Standards (NAAQS) for particulate matter 2.5 micrometers in diameter ($PM_{2.5}$) and smaller. These standards address long term (annual average) and short-term (24-hour average) concentrations of $PM_{2.5}$. Based on emerging data, it appears likely that many areas of the US will approach or exceed the annual-average threshold for compliance with the annual $PM_{2.5}$ NAAQS.

The health-based particulate matter standards will be reconsidered by EPA in 2002-2003, based upon a review of new health information.

The Inception of ARIES

The mandated timeline for the review process meant that EPA decisions would significantly benefit from new high-quality data on $PM_{2.5}$ mass and composition. This signaled the need for a collaborative effort between the public and private sectors to facilitate and expedite relevant data acquisition. In January 1998, Southern Company and EPRI formed a consortium of sponsors and eminent researchers to undertake a state-of-the-art air quality, health, and epidemiology study which promises to provide integral scientific input into the regulatory and standard-setting process in the years 2000 and beyond.

Measurements from the Southeastern Aerosol Research and Characterization study (SEARCH) and the convergence of a variety of complementary studies provided the baseline infrastructure for launching such a comprehensive study in Atlanta. After intensive planning and peer-review, investigators initiated ARIES air quality monitoring and health data collection in August 1998.

Objectives

The objective of ARIES is to investigate (via epidemiology and exposure studies) associations between air quality and human health and produce results in time for

consideration of the health basis of the NAAQS and for subsequent development of State Implementation Plans (SIPs). Fine PM may be an indicator (but not necessarily a cause) of adverse effects associated with inhalation — other pollutants, or PM components that co-vary with PM, may be the underlying cause. What sets ARIES apart from prior studies is that its focus is not on PM alone but on an unprecedented range of potential agents in the air, including VOCs, aeroallergens, and specific PM components. This comprehensive sampling approach enables a more robust and explicit investigation of the relationship between human health and airborne pollutants.

The study is fundamental by design, so that the results in terms of association between air quality and health will be generic and not limited to Atlanta. It is expected that the study will generate hypotheses to be tested through mechanistic studies and will provide data to test the relevance of results from toxicology studies. In short, ARIES will provide the underpinning of health-related studies for years to come.

Scope

ARIES is a multi-faceted study in which the disciplines of atmospheric research, epidemiology, exposure assessment, health assessment, and modeling were considered as parts of the whole from the inception of study design (see Figure 2). A comprehensive daily monitoring program will provide epidemiologists with a characterization of aerosol (gas and particle) physical, chemical, and biological (aeroallergenic) properties that has not been available to them before. There are four components of ARIES:

- *Air Quality Characterization:* $PM_{2.5}$ mass and composition, as well as related gas-phase and particle-phase pollutants, are measured every day at the Jefferson Street core monitoring station shown in Figure 1 with at least 24-hour time resolution. The air quality field measurements include SO_2, CO, NO, NO_2, NO_y, O_3, HNO_3, NH_3, and VOCs in the gas phase; major ions, including acidity, elemental/organic carbon (EC/OC), elements, water-soluble transition metals, and solvent-extractable carbon in the particle phase; pollen and mold; and particle number and size distribution from nanometers to micrometers in diameter.
- *Air Pollution Mortality:* daily mortality data are being collected and analyzed in a multi-pollutant ecological time-series study.
- *Air Pollution Morbidity:* daily data on emergency room (ER) visits are collected from practically all hospitals in the Atlanta area. The focus is on ER visits for coronary and respiratory symptoms. A parallel study is also being conducted to understand the influence of daily air quality on unscheduled physician visits at a large health-maintenance organization. Finally, the health study will evaluate the physiologic responses of a group of patients with more severe cardiac conditions (those with implanted defibrillators).
- *Exposure & Health Assessment:* a personal/indoor/outdoor exposure assessment study will help the epidemiologists assess how well ambient measurements can represent personal exposures for groups of individuals with recent heart attacks and with chronic obstructive pulmonary disease. This information may also have applications in validation of personal exposure models. The health study will also examine any association between exposure and cardiac response for these

participants. The representativeness of ARIES core site is also being assessed through specialized studies of spatial variability within Atlanta in ASACA.

Schedule

The first phase, including the full suite of data collection, began in July 1998 and ended in August 2000. ARIES is therefore one of the few studies that will be in a position to provide valuable new monitoring and health data in time for EPA's review of the PM standard. To add statistical power, most of the air quality and much of the health data collection are planned to continue through 2003

Collaborations

EPA selected Atlanta as its first designated 'Supersite' and conducted an intensive one-month experiment at the ARIES site in August 1999. The cooperative structure of ARIES allows for and fosters collaboration with EPA in integrating these private and public sector experiments.

Results to Date

Several scientific papers describing ARIES research findings have been published. These are listed below, and detailed findings can be found in the papers. Highlights from published findings include:

- Detailed characterization of $PM_{2.5}$ demonstrates the importance of carbonaceous matter;
- There are discrete episodes of elevated ultrafine particle numbers that are believed to be tied to anthropogenic emissions;
- $PM_{2.5}$ composition varies from hour-to-hour, day-to-day, and season-to-season. Sulfate comprises the largest fraction of $PM_{2.5}$ in summer, while carbonaceous matter comprises the largest fraction in the spring, fall, and winter;
- To date there are no reported statistically significant associations between deaths in Atlanta and any air quality variable; these results are based upon the first year of data; increased observations could change this result;
- Morbidity results are presently available only for hospital emergency room admissions data. To date no air quality variable has been associated with increased asthma or chronic obstructive pulmonary disease (COPD) admissions. Increased cardiovascular admissions have been associated with several air quality variables: carbon monoxide (CO); $PM_{2.5}$ elemental carbon (EC); $PM_{2.5}$ organic carbon (OC); and PM_{coarse} mass (i.e., the "coarse" fraction of PM_{10} or the difference between PM_{10} and $PM_{2.5}$).

ARIES at a Glance

Component Funding (approximate distribution)

Air Quality	$3.5 MM
Exposure Assessment	$1.5 MM
Epidemiology	$4.0 MM

Ambient Measurements (August 1998-August 2000)

Continuous (1-minute averages) O_3, NO, NO_2, NOy, HNO_3, SO_2, CO, $PM_{2.5}$ mass, wind speed, wind direction, temperature, relative humidity, barometric pressure, solar radiation, precipitation

Continuous (10-minute average)
Particle-count (0.003 to 3.0 μm)

Discrete (24-hour average)
$PM_{2.5}$ mass, ions, water-soluble metals, trace elements, organic carbon, elemental carbon
PM_{coarse} mass, ions, water-soluble metals, trace elements
Pollen and mold (>25 species)
VOCs, oxygenated VOCs and multi-phase VOCs
Ammonia

Fact Sheet

EPRI

Measurements by Collaborators
Single particle composition and size-resolved particle composition by NOAA
Continuous $PM_{2.5}$ mass and speciated monitoring at other sites by GIT (ASACA)
PM density (experimental) by University of Minnesota
EPA Supersite experiments

Sponsors
American Automobile Manufacturers Assoc.
American Electric Power Service Corp.
Alabama Electric Cooperative
Allegheny Energy
American Petroleum Institute
BG&E/Constellation
Central & South West Corp.
Connectiv
Detroit Edison
Duke Energy Corp.
Dynegy Midwest Generation
EPRI
First Energy Corp.
Great River Energy
LG&E Energy
Midwest Generation
Minnesota Power
National Rural Electric Cooperative Association
NiSource/NIPSCO
Oglethorpe Power Corp.
Reliant Energy HL&P
Salt River Project
South Carolina Electric & Gas Co.
Southern Company
Tennessee Valley Authority
TXU Electric
U.S. Department of Energy
Wisconsin Electric Power Co.
WPS Resources
Xcel

Collaborators
Southern Oxidants Study (SOS)
Southern Center for the Integrated Study of Secondary Air Pollutants (SCISSAP)
Southeast Aerosol Research Characterization Study (SEARCH)
Assessment of Spatial Aerosol Composition in Atlanta (ASACA)
EPA Supersite Program

Scientific Team
Atmospheric Research & Analysis
Desert Research Institute
EPRI
Emory University
Harvard University
Kaiser Permanente
Klemm Analysis Group
Oregon Health and Science University
University of Minnesota

Advisory Group
Tina Bahadori, American Chemistry Council
Rick Burnett, Health Canada
Isabella Romieu, Pan American Health Organization
Barbara Turpin, Rutgers University
John Vandenberg, US EPA
Warren White, Washington University

Published Papers
Woo, K. S.; Chen, D.-R.; Pui, D. Y. H.; McMurry, P. H. Measurements of Atlanta aerosol size distributions: observations of ultrafine particle events, *Aerosol Science and Technology*, **2000**, in press.

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Fact Sheet

EPRI

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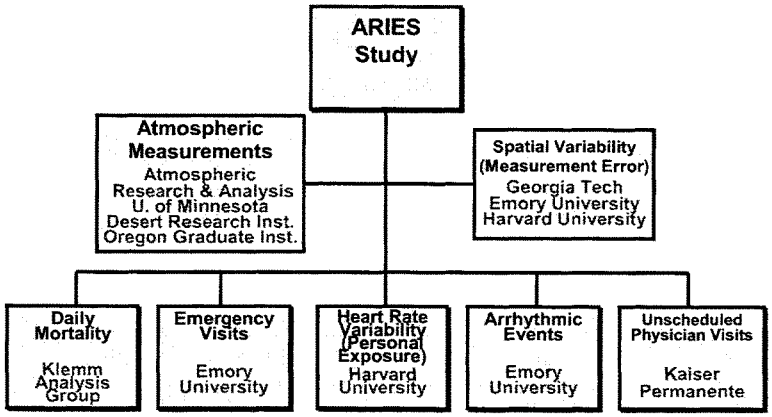


Figure 2. ARIES Components

STATEMENT OF ROBERT O'KEEFE, VICE PRESIDENT, HEALTH EFFECTS INSTITUTE

Mr. Chairman, members of the committee, it is a pleasure to have this chance to appear before you to share the perspective of the Health Effects Institute on what we have learned and what we still need to learn about the health effects of particulate matter. For the record, I am Robert O'Keefe, Vice President of the Health Effects Institute, an independent research institute funded jointly and equally by the US EPA and industry to provide impartial and high quality science on the health effects of air pollution.

THE DATA WE HAD IN 1997—SHORT AND LONG TERM EPIDEMIOLOGY

In 1997, the US EPA promulgated a new set of National Ambient Air Quality Standards (NAAQS) for fine particulate matter ($PM_{2.5}$). In large measure, that action was based on two types of epidemiology studies:

- There were nearly 40 short-term studies that found a statistical relationship between daily changes in air pollution and daily small but relatively consistent increases in daily levels of death, hospitalization, and illness (e.g. 1% to 2% increases in mortality for every 10 microgram/cubic meter increase in PM_{10});
- Two long-term "cohort" studies—the Harvard Six Cities Study and the Pope/American Cancer Society Study—that tracked selected populations of people in a series of more- and less-polluted cities, and found that those who lived in the most polluted cities had between a 17% and 26% higher risk of premature death than those who lived in the least polluted cities.

These studies suggested that a measurable portion of mortality and respiratory and cardiac illness in the United States might be attributable to fine particle air pollution, and based on them, EPA set the new, more stringent NAAQS for $PM_{2.5}$. At the same time, there were a number of questions about these studies, key among them:

- The individual short-term studies were done by diverse investigators using somewhat different methods—would a more systematic study find the same results?
- Could other pollutants, which occur along with $PM_{2.5}$, be more likely to be responsible for the increased mortality?
- Did the deaths measured in these short-term studies represent substantial losses of life years, or the advancing of death for critically ill people by a few days?
- Did the exposures measured in these studies—at central air pollution monitors—accurately represent the exposures of people who in general spend most of their time indoors?
- Could the Harvard Six Cities Study and the American Cancer Society Study, whose data had only been analyzed by the original investigators, stand up to intensive scrutiny and analysis from new, independent investigators? Could there be other differences between the cities (e.g. differences in socioeconomic status or health care) that would also explain the differences in mortality?

In addition to these questions about the epidemiology, there were also questions about the relative toxicity of the many different components of the complex PM mixture, and about the possible biological mechanisms that might explain the epidemiology results, questions that were laid out in a 1998 priority research agenda by the National Academy of Sciences Committee on Research Priorities for Airborne Particulate Matter.

WHAT HAVE WE LEARNED SINCE 1997?

Since 1997, substantial new research has been undertaken to advance our understanding of the health effects of PM. As one part of the larger effort undertaken, HEI has invested in some 40 epidemiology, exposure, and toxicology studies to test the validity of the original studies, and to begin to answer the remaining questions.

Key among HEI's work have been two efforts to determine the validity of the short- and long-term epidemiology studies—the National Morbidity, Mortality, and Air Pollution Study (or NMMAPS), and the Reanalysis of the Harvard Six Cities and American Cancer Society studies.

The National Morbidity, Mortality, and Air Pollution Study (or NMMAPS)

NMMAPS is a systematic study of air pollution, weather and mortality in the 90 largest cities in the United States, conducted—under the oversight, quality assurance procedures, and review of HEI—by investigators at Johns Hopkins University. NMMAPS also included similar analyses of air pollution and elderly hospitalization, conducted in 14 U.S. cities by investigators at Harvard University.

In brief, this systematic and rigorous study found a consistent relationship between PM_{10} and mortality in the 90 largest cities of an approximately 0.4% increase in mortality for every 10 micrograms increase in PM_{10}. This level of effect was about

half the size of that found in the earlier study, but as the graph in my testimony illustrates, this effect was not substantially affected by any of the other gaseous air pollutants. (See Figure 1) The NMMAPS investigators also found that at least a portion of the mortality was not solely frail people dying a few days early, but deaths advanced 30 days or more, and conducted analyses that suggested that errors from using centrally-monitored air pollution to estimate exposure were not likely to change the basic results.

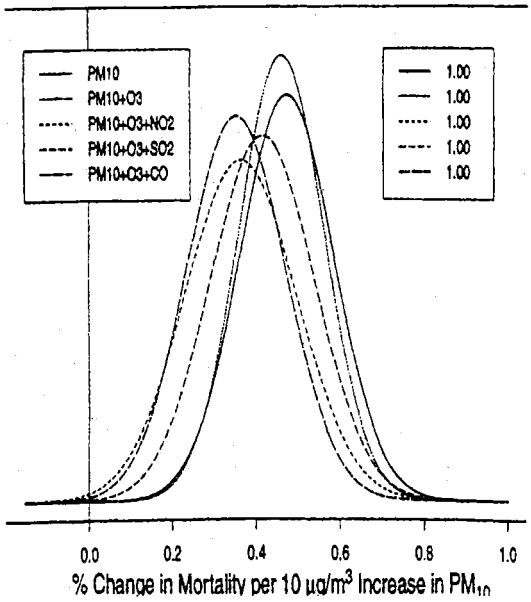


Fig.1 NMMAPS Analyses of PM vs. Other Pollutants

At the same time, this first nationwide analysis found differences in levels of effect across the U.S., suggesting that other factors, perhaps different mixes of pollution, could contribute along with particles to the effect. (See Figure 2) Overall, the NMMAPS analyses provided greater confidence in the results of the short-term epidemiology.

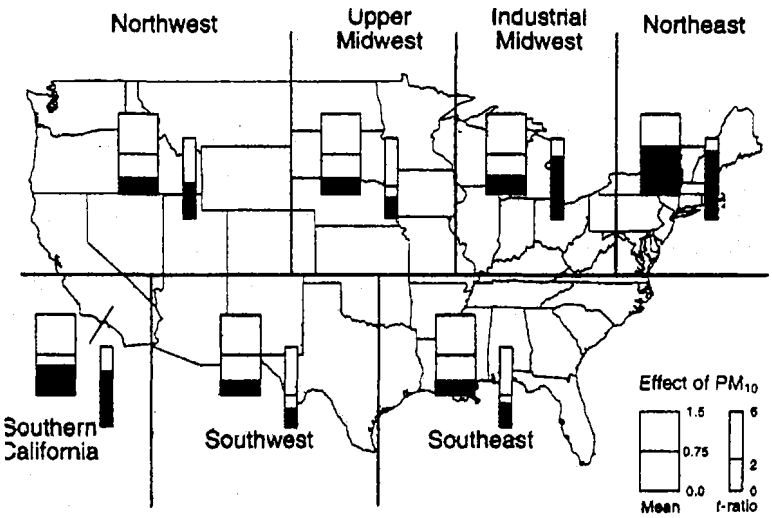


Fig.2 NMMAPS Regional Analyses

NMMAPS Update: This past Spring, members of the original team of investigators from the Johns Hopkins School of Public Health were conducting additional analysis on their findings of an association between daily changes in air pollution and mortality. In the course of testing these analyses against different assumptions and examining the methods used, they identified a generally unknown aspect of S-Plus, a statistical software package widely used by air pollution and other investigators to fit general additive models (GAMs) to data. In NMMAPS the investigators found that the result of using this approach was to overstate the effect estimates in this study. Upon notification of these new findings, HEI mobilized its NMMAPS Review Panel, Chaired by Dr. Sverre Vedal of the National Jewish Medical and Research Center in Denver. The panel provided initial peer review of the work of the investigators to apply alternative analytic techniques to the data to correct for this effect. In brief the Panel found that:

- most of the raw, unadjusted individual city estimates changed, with an increased number of estimates that were negative or zero;
- the mean effect estimate shifted from .41 increase in mortality for every 10 micrograms increase in PM_{10} (in the original study) to .21 percent (in the revised analysis);
- in the revised results, PM_{10} effect estimates are unaffected by the addition of co-pollutants such as ozone. (see Figure 3 below)

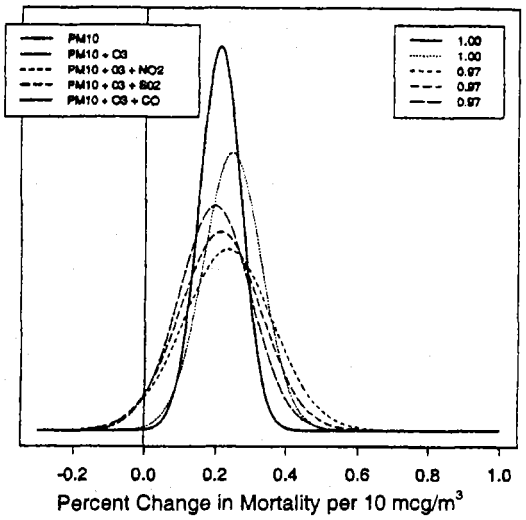


Fig. 3 Revised NMMAPS Analysis of PM vs. Other Pollutants

The HEI Panel continues to review the work of the investigator teams from both Johns Hopkins and Harvard to recalculate key analyses in the studies and provide comprehensive HEI peer review and commentary. A final report is expected in January. In addition, a number of other studies cited in EPA's current draft Criteria Document for Particulate Matter also use this software and may be affected in similar or different ways. To assess the nature and extent of this effect, US EPA and its Clean Air Scientific Advisory Committee (CASAC) at its July meeting set out a multi-step process to identify studies central to the NAAQS and recalculate key analyses in these studies. HEI, at the request of EPA and CASAC will play a central role in the review of these analyses.

The Reanalysis of the Harvard Six Cities and American Cancer Society Studies

In addition to NMMAPS, and in response to requests from Congress, US EPA, industry and others, HEI convened a detailed reanalysis of the Harvard Six Cities and American Cancer Society studies. Given full access to the entire medical and air pollution data base from the original investigators, HEI's Expert Panel selected an entirely new team of investigators, conducted a detailed quality assurance audit of the data and replication analyses, and then implemented a large number of sensitivity analyses to test whether some other difference between the most and least polluted cities (e.g. differences in the quality of medical care) could explain the increased mortality risk.

In brief, the reanalysis assured the quality of the data, replicated the original results, and tested those results against alternative risk models and analytical approaches without substantively altering the original findings of an association between sulfates (a form of particles created in the atmosphere from coal combustion and other emissions) and fine particles ($PM_{2.5}$) and mortality (see Table 1 below).

Table 1.—Relative Risk of Mortality for Those Living in Most Polluted City in ACS Study for Original Analysis and Reanalyses

[E.G., in original analysis those living in city with the highest $PM_{2.5}$ had a 17% higher risk of mortality]

Analysis	$PM_{2.5}$	Sulfates
Original	1.17 (1.08,1.27)	1.15 (1.08,1.22)
Full	1.18 (1.09,1.26)	1.15 (1.09,1.21)
Extended	1.18 (1.09,1.26)	1.15 (1.09,1.21)

At the same time, the reanalyses extended and challenged our understanding of the original results:

- the effects on mortality appeared to increase for those with less education (and likely therefore of lower socioeconomic status;
- when the correlations among cities near one another were considered, the effects of fine particles remained but were diminished; and
- an association between sulfur dioxide (SO_2) and mortality (but not other pollutants) was observed and persisted when other variables were included.

In conclusion: the reanalysis identified relatively robust associations of mortality with fine particles, sulfate, and sulfur dioxide, and tested those associations in nearly every possible manner within the limitations of the data sets.

KEY QUESTION FOR THE LONGER TERM: ARE ALL PARTICLES CREATED EQUAL?

To date, most analyses of the effects of particulate matter have focused on the mass of PM. Particles are, however, a complex mixture of pollutants, and over the longer term, it will be important to understand whether all particles have similar levels of toxicity, or whether some particles, and therefore some sources, contribute higher toxicity, and should be more stringently controlled. While there are many actions underway already to reduce overall particle levels—for example to control diesel vehicle PM emissions and nitrogen oxide emissions (a precursor of nitrates) from power plants—in the years to come, it will be especially important to develop the most cost-effective control strategies aimed at the most toxic sources, or at the most toxic components of those sources' emissions. This will be a critical area for new research.

There are a number of components of PM that could cause toxicity. At a multidisciplinary NARSTO/EPA workshop in July, 1998, the following key PM characteristics and components were identified:

- PM mass
- PM particle size, surface area
- Ultra fine PM
- Reactive transition metals
- Organic compounds (e.g. diesel PM)
- Acids
- Biogenic particles
- Sulfates and nitrates (e.g. from SO_2 and NOx)
- Peroxides
- Soot
- Co-pollutants—SO_2, CO, Ozone, etc.

Research studies are now underway at EPA, HEI, EPRI, NIEHS, and other research institutions to begin to identify the relative toxicity of some of these components. Initial indication of the potency of some of these elements (e.g. the metals attached to PM) are beginning to emerge. In some cases, studies have looked at effects of emissions from power plants. Some studies have not found effects from exposure to sulfates; however other studies, including the reanalysis and toxicology studies, have found effects of sulfates and other potential emissions such as fly ash. Ultimately, identifying whether one or more of these components is especially toxic will require a systematic, multidisciplinary effort.

To address these questions, the HEI Review Committee, in April 2002, issued the second in its HEI Perspectives series entitled, "Understanding the Health Effects of Components of the Particulate Matter Mix: Progress and Next Steps." This review, which I have provided to your staff and is available on the HEI web site at *http://www.healtheffects.org/Pubs/Perspectives-2.pdf*, summarizes recent HEI and other research on the effects of different components of the mix. It also lays out a systematic effort necessary to achieve a better understanding, including:

- Parallel epidemiology studies in carefully selected, representative cities throughout the U.S., with detailed daily characterization of the particle mixture;
- Companion toxicology studies using concentrated ambient particles, source-specific particles, and model particles to test the full range of health endpoints and mechanisms for each particle type.

Many elements of such an effort are currently underway in the EPA research program and other efforts. A more systematic approach will require substantial resources dedicated over the next decade. However, the result of such an effort could be a better-focused and more cost-effective path to improved public health.

CONCLUSION: PROGRESS AND NEXT STEPS

In conclusion, we have made much progress in the last five years, especially in testing the validity of the short- and long-term epidemiology studies which served

as the primary basis for the setting of the 1997 NAAQS for particulate matter. We have tested a number of possible confounding factors, explored whether errors in measuring exposure might explain the relationships between PM and health, and analyzed whether different statistical techniques could change the results. In reviewing the latest evidence, the HEI Review Committee concluded "epidemiologic evidence of PM's effects on mortality and morbidity persists even when alternative explanations have been largely addressed". Based on this evidence, a number of initial control measures are now moving forward.

At the same time, important new questions have arisen. In the near term it is necessary to complete the reassessment of NMMAPS and identify, reassess and provide peer review for other key studies that use GAM. Over the longer term, other important questions also remain, especially concerning the comparative toxicity of different components and sources of the PM mixture. Much research is underway to understand this important question and to inform and target future strategies for control of those emissions that may be most responsible. Only through a systematic effort to test and compare the toxicity of these diverse particles will we be able to have the best chance of answering these key questions for the future.

Thank you again for the opportunity to present this testimony. I would be pleased to answer any questions you might have.

STATEMENT OF JONATHAN LEVY, ASSISTANT PROFESSOR, ENVIRONMENTAL HEALTH AND RISK ASSESMENT, DEPARTMENT OF ENVIRONMENTAL HEALTH, HARVARD SCHOOL OF PUBLIC HEALTH

The materials included in this written testimony provide support for my oral presentation regarding the implications of the $PM_{2.5}$ health literature for power plant risk calculations.

In my oral testimony, I focused on the evidence for mortality risks from particulate matter, given the important role that mortality has played in past benefits assessments of air pollution controls (such as the EPA's benefit-cost analysis of the Clean Air Act). I also asserted that there are three crucial questions that must be answered to quantify the public health benefits of power plant pollution controls:

1. Is there a threshold below which no health effects of $PM_{2.5}$ are found, and if so, where is that threshold?
2. Do all types of particulate matter have similar health impacts, or are some particles more toxic than others?
3. Would alternative control strategies have significant impacts on the magnitude or distribution of particulate matter health impacts?

Within this document, I address these three questions in greater detail, summarizing the key studies that inform my answers to these questions. Along with this summary document, I have included copies of selected documents that provide even more information about the core issues.

IS THERE A THRESHOLD?

An initial point that is important to emphasize is that this is not the same question as whether $PM_{2.5}$ concentrations are above or below National Ambient Air Quality Standards. Quoting directly from the US EPA in their Final Rule for the $PM_{2.5}$ NAAQS, "The Act does not require the Administrator to establish a primary NAAQS at a zero-risk level, but rather at a level that reduces risk sufficiently so as to protect public health with an adequate margin of safety." (p. 3). The question is therefore whether the health literature provides evidence of a threshold above current ambient concentrations.

First considering time-series studies, which evaluate the effects of changes in daily concentrations of PM on daily mortality risks, two major studies illustrate the nature of the literature (Daniels et al., 2000; Schwartz et al., 2002). The first of these studies used information from the National Morbidity, Mortality, and Air Pollution Study (NMMAPS) to evaluate whether a threshold existed for short-term exposure to PM_{10}, either for total mortality or cardiovascular/respiratory mortality. The authors showed that for daily changes in PM_{10}, linear models without thresholds were most appropriate for total or cardiovascular/respiratory mortality. When considered probabilistically, the threshold for total daily mortality appeared to be definitely below 30 $\mu g/m^3$ and was most likely below 15 $\mu g/m^3$. The second study used information from the Six Cities Study, considering daily mortality risks from changes in $PM_{2.5}$ concentrations. As with the NMMAPS study, the authors concluded that a linear no-threshold model was most appropriate.

Thresholds have also been examined in the cohort mortality literature, with the most recent evidence provided in the follow-up to the American Cancer Society co-

hort study (Pope et al., 2002). Within the range of concentrations in the study, there was no evidence of a threshold, and the relationship appeared approximately linear. The lowest concentrations reported in the study (averaged across the study period) were less than 10 $\mu g/m^3$.

Thus, the epidemiological literature shows no evidence of a threshold for mortality risks at current ambient concentrations. Although this may be counter-intuitive, given the normal assumptions regarding thresholds for non-carcinogens, this relationship is biologically plausible. As explained in Schwartz et al. (2002), individuals will likely have thresholds, but if those thresholds differ widely across individuals based on numerous factors, then the distribution of thresholds across the population should be normally distributed. This would imply that the population concentration-response curve would approximately a cumulative normal curve, which is linear at low concentrations. In other words, if current particle levels were below the mortality threshold for most (but not all) people, then linearity with no population threshold would be expected.

DO ALL TYPES OF PARTICLES HAVE SIMILAR HEALTH EFFECTS?

Prior to evaluating the literature, it is important to frame this question appropriately. Because most of the epidemiological evidence available to date has been based on monitors that measure total particulate mass in various size ranges, it has been established that particulate matter concentrations are associated with mortality and morbidity. However, little information has been available about the relative toxicity of different types of particles, so the default assumption has been that all pollutants have equal toxicity.

While that is unlikely to be the case, to deviate from this assumption, one must be able to quantify relative toxicities and defend these quantifications. Explicitly, for the case of power plant emissions, we would need to be able to estimate how toxic a sulfate or nitrate particle is relative to average particles. Clearly, this is not a question that can be answered with certainty, nor is it one that will be definitively solved in the near term.

Focusing on epidemiological evidence, there are two types of studies available: studies that directly measured at least one of the constituents of interest (often sulfates) and studies that used statistical methods to try to determine source-specific differential toxicity. Each approach has advantages and limitations, and each can add to the body of evidence.

In cohort mortality investigations, the primary evidence arises through the analysis of sulfates along with particulate mass in various size fractions. In the Harvard Six Cities Study (Dockey et al., 1993) and American Cancer Society study (Pope et al., 2002), long-term exposure to sulfates displayed a consistent positive association with premature mortality. In the latter publication, as well as in the Health Effects Institute reanalysis (Krewski et al., 2000), the authors concluded that some combination of $PM_{2.5}$, sulfates, and possibly SO_2 were associated with mortality. In a third cohort study (McDonnell et al., 2000), sulfates were not statistically significant, although the central estimate for mortality for male nonsmokers from sulfates was between the values from the Six Cities and American Cancer Society studies.

In terms of the relative effect of sulfate versus general $PM_{2.5}$, our power plant risk assessment in Massachusetts (Levy and Spengler, 2002) found that impacts were greater if either the reported sulfate-mortality or SO_2-mortality relationship were applied rather than the $PM_{2.5}$-mortality relationship. Thus, the cohort mortality literature generally shows sulfate effects that are significant, with a concentration-response function slightly greater than general $PM_{2.5}$ effects and no direct information available on other particulate species.

In the time-series literature, much of the speciation data come from studies looking at sulfates. These studies have generally found positive associations, as indicated in the following figure (taken from the second external review draft of the Particulate Matter Criteria Document).

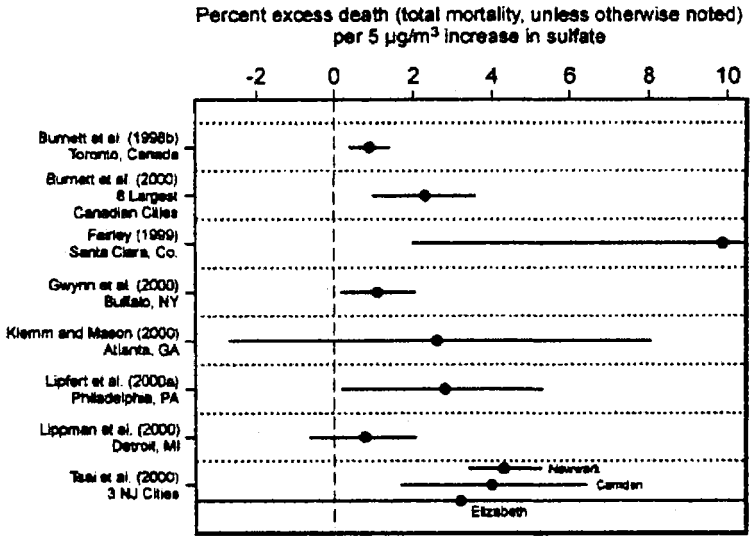


Figure 6-5. Excess risks estimated for sulfate per 5 $\mu g/m^3$ increase from the studies in which both $PM_{2.5}$ and $PM_{10-2.5}$ data were available.

As indicated in the above figure, there has been preliminary evidence available from the supersite in Atlanta, which measures numerous chemical species (Klemm and Mason, 2000). This study found no statistically significant relationship for any particulate measures using one year of time-series data. Per unit concentration, the central estimates were higher for elemental carbon and sulfates than for $PM_{2.5}$ as a whole, with lower central estimates for organic carbon and nitrates (although no values were statistically significant). In interpreting these results, it is important to realize that lack of statistical significance could be related to either a lack of an effect or a lack of statistical power to find an effect, given a relatively small sample size. If we look at the body of sulfate time-series studies in the above figure, we see that the Klemm and Mason findings in fact have a central estimate in line with much of the previous literature, but with substantially wider confidence intervals. Once this study is completed, it should be combined with other available studies to determine a best estimate for the time-series relationship between sulfates and mortality, taking into account relevant site and population characteristics (e.g., air conditioning prevalence) to generalize to the U.S. at large.

Looking at studies of source-specific effects, a study by Laden and colleagues (2000) applied statistical methods to elemental data from the Six Cities study to determine source-specific particulate matter factors. Across all six cities, they found that the motor vehicle and coal factors had statistically significant effects on premature mortality, with the motor vehicle factor approximately a factor of three greater than the coal factor (per unit concentration). A crustal factor was not significant. Although the confidence intervals were wide, there was some evidence that cardiovascular deaths were more closely related to motor vehicle particles and respiratory deaths were more closely related to coal-derived particles.

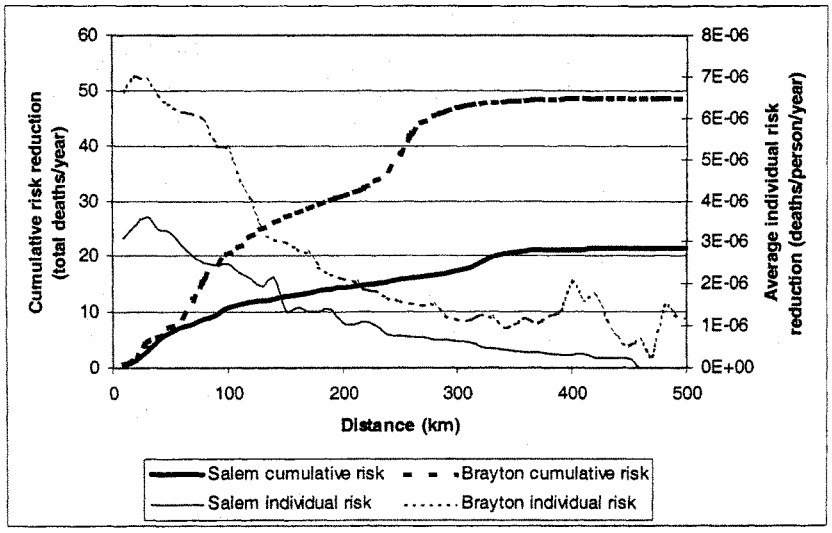
Additional factor—analytic studies include Ozkaynak and Thurston (1987) and Mar (2000). In the former study, based on cross—sectional mortality data across the U.S., particles from industrial sources and coal combustion had greater coefficients than those from motor vehicles or crustal sources. In the latter study in Phoenix, combustion-related pollutants (from motor vehicles and vegetative sources) and secondary sulfates were associated with cardiovascular mortality. A soil-related factor had a negative association with mortality. Thus, the findings from factor analytic studies appear to show lower toxicity of crustal particles, with significant effects from motor vehicles, power plants, and other combustion sources. However, the studies do not provide consistent quantitative evidence for greater toxicity of one combustion source category over another.

In conclusion, while it is difficult to assign specific differential toxicities to different particle types, it does appear likely that combustion particles are more toxic than crustal particles. In studies looking at both sulfates and PM, the effect per unit concentration of sulfates is generally slightly higher, but the relatively small difference and the lack of substantial toxicological evidence makes a conclusion of equal toxicity reasonable as a central estimate for risk calculations.

WHAT ARE THE MAGNITUDE AND DISTRIBUTION OF PM HEALTH EFFECTS FROM POWER PLANTS?

First considering the distributional question, it is clear that the impacts from a single power plant will vary spatially (since the concentrations associated with that plant will not be uniform across the country). The crucial question is whether populations near the power plants are disproportionately at risk or whether the impacts occur at longer distances, as this will influence the formulation of optimal control strategies.

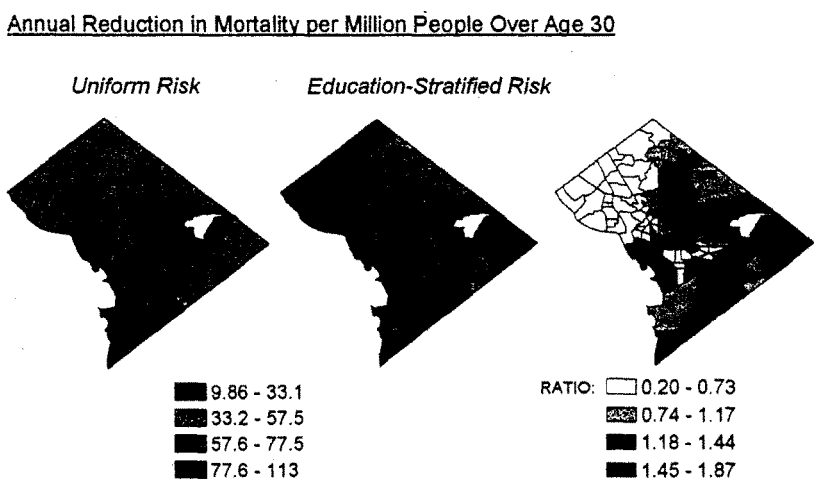
In our initial power plant analysis in Massachusetts (Levy and Spengler, 2002), we concluded that the answer to this question depended largely on how the question was framed. We distinguished between individual risk (the mortality risk to a given individual at a given location) and aggregate risk (the total public health impact associated with the facility). When we look at individual risk, the maximum occurs relatively close to the power plants—approximately 25–40 km away for the two plants studied in Massachusetts. However, because of the long-range transport of particulate matter and the number of people who are impacted at long range, most of the aggregate risk occurs at long range—more than half beyond 100 km, as illustrated in the figure below from Levy and Spengler (2002). Thus, we can conclude that individuals who live closer to a power plant are more impacted by that plant than individuals living further away, but that local populations contribute a relatively small fraction of aggregate risk.



Although this captures broad distributional trends related to distance from the source, another aspect of the distributional question is whether selected demographic groups are disproportionately affected by power plant air pollution. If this is the case, then a greater amount of the population risk occurs in a smaller set of individuals, which increases the importance of considering distributional issues.

In a recent power plant risk assessment focused on the Washington, DC area (Levy et al., in press), we identified high-risk populations for selected health outcomes and evaluated the implications for the magnitude and distribution of health benefits. For the case of premature mortality, we considered the influence of educational attainment on mortality risk, as documented in Pope et al. (2002). We concluded that if the observational evidence from the American Cancer Society cohort

study were correct, then more than half of the health benefits accrued among the 25% of the population with less than high school education. Furthermore, we showed that small-scale spatial variations were significantly influenced by the incorporation of population patterns, as illustrated by Figure 4 from Levy et al. (in press), a portion of which is reproduced on this page.



Finally, I turn to the question of estimating the magnitude of health impacts from power plant emissions. Making this estimate requires a multi-step process. First, the emissions of SO_2 and NOx are quantified (given the structure of multi-pollutant regulations and the focus on particulate matter impacts). Second, atmospheric dispersion models are used to evaluate the influence of these emissions on concentrations of $PM_{2.5}$ across a large region. These concentration changes are then combined with epidemiological evidence to quantify the public health implications.

As an example of this sort of analysis, Abt Associates (2000) used an economic model to estimate the distribution of SO_2 and NOx emissions from the power sector given proposed emission controls, applied two atmospheric dispersion models to evaluate the national $PM_{2.5}$ implications of these proposals, and linked the concentration changes with health evidence, including the mortality risk derived from the American Cancer Society cohort study. They concluded that current power plant emissions were associated with approximately 30,000 premature deaths per year, with a 75% reduction scenario yielding benefits of approximately 19,000 fewer premature deaths per year.

A critical question is whether these estimates represent reasonable central estimates or are biased in either direction. In a recent investigation (Levy, 2002), I reviewed the methodology used by Abt Associates in a similar analysis, focusing on the question of bias. I considered separately the atmospheric model and the health evidence. I concluded that the atmospheric model yielded health impact estimates that were essentially identical to those using a different model, and that the concentration-response function chosen for premature mortality was a reasonable central estimate. Thus, it appeared equally likely that the Abt Associates methodology yielded an underestimate as an overestimate, making their findings a reasonable foundation for policy decisions.

A similar methodology was used by the EPA to estimate the benefits of alternative power plant control policies. For example, the EPA estimated that the Clear Skies Act would reduce premature deaths by about 12,000 per year, by combining the results of atmospheric models and epidemiological studies (see *www.epa.gov/clearskies*). Similarly, an earlier straw proposal from the EPA (which had more stringent caps on both SO_2 and NOx) was associated with a reduction of 19,000 premature deaths per year. Again, this was based on a similar methodology as used by Abt Associates, implying that the estimate is a reasonable central estimate.

From the above discussion, it is qualitatively clear that increased reductions of SO_2 and NOx are likely to lead to increased public health benefits. While the above public health estimates are clearly uncertain, they appear just as likely to be underestimates as overestimates. Thus, it is reasonable to assume that the Clear Skies

Act would provide substantial public health benefits, but that the EPA straw proposal (which is similar to the Clean Power Act) would increase those benefits by perhaps 7,000 fewer premature deaths per year. This implies that choices between status quo emissions, the Clear Skies Act, the Clean Power Act, and other alternative formulations should depend on a comparison of the incremental costs and benefits of increased stringency.

ATTACHED DOCUMENTS

I have attached a subset of the studies cited above, which either expand on the arguments in this testimony or are not yet publicly available. Attached documents include:

- Levy J. Evaluation of Methodology in "Particulate-Related Health Impacts of Eight Electric Utility Systems". Prepared for Rockefeller Family Fund, June 2002.
- Levy JI, Greco SL, Spengler JD. The importance of population susceptibility for air pollution risk assessment: A case study of power plants near Washington, DC. Environ Health Perspect, in press, December 2002 expected.
- Levy JI, Spengler JD. Modeling the benefits of power plant emission controls in Massachusetts. J Air Waste Manage Assoc 52: 5–18 (2002).
- Pope CA III, Burnett RT, Thun MJ, Calle EE, Krewski D, Ito K, Thurston GD. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. JAMA 287:1132–1141 (2002).
- Schwartz J, Laden F, Zanobetti A. The concentration-response relationship between $PM_{2.5}$ and daily deaths. Environ Health Perspect 110: 1025–1029 (2002).

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Daniels MJ, Dominici F, Samet JM, Zeger SL. Estimating particulate matter-mortality dose-response curves and threshold levels: an analysis of daily time-series for the 20 largest U.S. cities. Am J Epidemiol 52: 397–406 (2000).

Dockery DW, Pope CA III, Xu X, Spengler JD, Ware JH, Fay ME, Ferris BG. Jr., Speizer FE. An association between air pollution and mortality in six U.S. cities. N Engl J Med 329: 1753–1759 (1993).

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Krewski D, Burnett RT, Goldberg MS, Hoover K, Siemiatycki J, Jerrett M, Abrahamowicz M, White WH. Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality. Cambridge, MA: Health Effects Institute (2000).

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Levy JI, Greco SL, Spengler JD. The importance of population susceptibility for air pollution risk assessment: A case study of power plants near Washington, DC. Environ Health Perspect, in press, December 2002 expected.

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Ozkaynak H, Thurston GD. Associations between 1980 U.S. mortality rates and alternative measures of airborne particle concentration. Risk Anal. 7: 449–461 (1987).

Pope CA III, Burnett RT, Thun MJ, Calle EE, Krewski D, Ito K, Thurston GD. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. JAMA 287: 1132–1141 (2002).

Schwartz J, Laden F, Zanobetti A. The concentration-response relationship between $PM_{2.5}$ and daily deaths. Environ Health Perspect 110: 1025–1029 (2002).

June 20, 2002

Eric Schaeffer
Rockefeller Family Fund
437 Madison Ave., 37th floor
New York, NY 10022

Dear Eric:

Following is a report that provides a detailed and quantitative examination of the models and assumptions used in the Abt Associates study titled "Particulate-Related Health Impacts of Eight Electric Utility Systems". This analysis represents a follow-up of my initial letter dated May 20, 2002.

The key conclusions from my analysis are as follows:

- The atmospheric model applied appropriately accounts for critical atmospheric factors and provides health impact estimates quite similar to estimates from other models.
- The health evidence considered includes the major published studies on particulate matter health effects. The concentration-response function for mortality is a reasonable interpretation of the current literature, and the evidence cited for other health outcomes is representative of the literature as a whole.

Based on the calculations provided, I conclude that the health estimates in the Abt Associates study are well supported by the published literature and represent reasonable central estimates. Substantial bias related to the atmospheric modeling is extremely unlikely, and bias related to the health evidence (in either direction) is only plausible only under extreme interpretations of the literature. I therefore conclude that the Abt Associates study provides useful information from which policy conclusions can be drawn, and information as provided in the attached document can be used to provide plausible upper and lower bounds for the public health impacts of selected power plants.

Jonathan Levy

Evaluation of Methodology in "Particulate-Related Health Impacts of Eight Electric Utility Systems"

Executive Summary

In April 2002, Abt Associates prepared a report titled "Particulate-Related Health Impacts of Eight Electric Utility Systems". In this report, the authors focused on quantifying the health impacts of particulate air pollution from power plants in eight electric utility systems (AEP, Cinergy, Duke, Dynergy, First Energy, SIGECO, Southern, and TVA). They concluded that this subset of power plants could be associated with 5,900 premature deaths per year, 140,000 asthma attacks per year, and 6,000,000 minor restricted activity days per year, among other health outcomes. These estimates were made by forecasting NOx and SO_2 emissions at the selected power plants in 2007, using a relatively simple atmospheric dispersion model to estimate the resulting particulate matter impacts across the US, and using current health evidence to quantify the health impacts from this incremental contribution to particulate matter levels.

In any analysis of this sort, there are numerous uncertainties, many of which are impossible to eliminate. The important question for policy analysis is whether the estimates in this report are significantly biased (in either direction), how large the uncertainties appear to be, and what the major contributors to uncertainty are.

In this review, I focus on the questions of bias related to the atmospheric dispersion model and the use of health evidence. Through detailed comparisons with other similar studies using more complex atmospheric models, I conclude that the atmospheric dispersion model in the Abt Associates report does not appear to be significantly biased. Nitrate impacts are likely more uncertain than sulfate impacts, because of issues related to the atmospheric chemistry, but

this would have a relatively small impact on the total health impacts due to the far greater contribution of sulfates.

Similarly, the decisions regarding relative toxicity of particle types and the choice of studies made by the authors appear appropriate and provide reasonable best estimates of health impacts. Although alternative interpretations of the health literature are available and could lead to significantly different estimates, the concentration-response functions selected by Abt Associates are bounded by estimates available elsewhere and are reflective of current scientific knowledge. The most significant uncertainty is related to the interpretation of cohort mortality studies, but given currently available information, it would be inappropriate to exclude this effect entirely from a comprehensive analysis.

The estimates provided in the text of my review can help decision makers place upper and lower bounds on the potential magnitude of the health effects and determine in which areas further research might help inform policy decisions. In conclusion, supported by a detailed uncertainty analysis, the estimates from the Abt Associates report provide a plausible basis for near-term policy decisions.

Evaluation of Dispersion Model

For the atmospheric modeling, it is important to evaluate the potential biases or uncertainties in the model from a health-relevant perspective. In other words, it would be possible for S-R matrix to display different geographic patterns than other models, but result in similar health impact estimates. Since the Abt Associates report focuses largely on national health impacts, I evaluate S-R matrix from a national perspective as well. Some of the discussion below, including the comparison for power plants in Georgia, is based on the analysis in a manuscript in preparation (*1*).

To compare the results from different atmospheric models in a risk assessment and to allow for model results to be extrapolated to other settings, analysts have developed the concept of the intake fraction (*2*). An intake fraction can be defined simply as the fraction of a pollutant or its precursor emitted that is eventually inhaled or ingested by someone, somewhere. Mathematically, it is defined as:

$$iF = \frac{BR \times \sum_i C_i \times N_i}{Q}$$

where iF = intake fraction; BR = population-average breathing rate (assumed to be 20 m^3/day); C_i = incremental concentration of pollutant at receptor i ($\mu g/m^3$); N_i = number of people at receptor i; Q = emission rate of pollutant or pollutant precursor (μg/day).

Therefore, an intake fraction is a unitless measure that depends on how a pollutant emitted influences ambient concentrations, and on how many people are affected by those concentrations. If the health effects of the pollutant have a linear concentration-response function

with no dose rate dependence, this figure will be directly proportional to health impacts. In other words, if an intake fraction from Source A is double the intake fraction from Source B, then if the sources have the same emission rate, the health impacts from Source A will be double those of Source B. Since the Abt Associates report assumes linearity in concentration-response functions, the intake fraction is an appropriate figure to estimate.

For this case, we are exclusively interested in intake fractions related to particulate matter formation due to SO_2 and NOx emissions, as the Abt Associates report focused on health effects from particulate matter. Clearly, SO_2 emissions lead to the formation of ammonium sulfate particles and NOx emissions lead to the formation of ammonium nitrate particles. However, there is an additional intake fraction we must consider. It is a well-established fact that, under some conditions, changes in SO_2 emissions can influence particle nitrate concentrations. Because ammonium preferentially reacts with sulfate over nitrate, decreases in sulfate concentrations can potentially free up ammonium to react with nitrate. Thus, throughout this text, I will be considering three different intake fractions:

- Sulfate/SO_2: Incremental amount of sulfate inhaled per incremental unit of SO_2 emissions
- Nitrate/NOx: Incremental amount of nitrate inhaled per incremental unit of NOx emissions
- Nitrate/SO_2: Incremental amount of nitrate inhaled per incremental unit of SO_2 emissions

Given these definitions, the question is: Are the intake fractions implied by the S-R matrix analysis similar to those from other modeling studies? We address this question by making two major comparisons:

1. S-R matrix versus CALPUFF for 40 power plants randomly selected across the US (7 of which are in the Abt Associates report)
2. S-R matrix versus CALPUFF for seven power plants in Georgia (all included in the Abt Associates report)

For the first comparison, we are comparing the findings from Wolff (*3*) with findings from the S-R matrix, which was provided to us by Abt Associates. Wolff used CALPUFF to model the intake fractions for primary PM, sulfates, and nitrates for 40 power plants randomly selected across the US. CALPUFF is a transport and dispersion model that models emissions as a sequence of discrete puffs and simulates both dispersion and chemical transformation (*4*). It is generally applied to a small set of sources with limited background pollution data. For these and other reasons, CALPUFF has a somewhat different methodological framework than S-R matrix.

In Wolff (*3*), CALPUFF was used to estimate the incremental concentrations for each source at each of 448 receptor points, spaced every 100 km over a region 1600 km by 2800 km. To estimate intake fractions, Wolff used 1990 meteorological and population data, as taken from ArcView version 3.2. Of note, this implies that the intake fractions estimated by Wolff would be expected to be slightly lower than the intake fractions implied by S-R matrix, which use 2007 population data. The ratio between 2007 and 1990 US populations is roughly 1.17. In addition, CALPUFF default values of parameters such as particle size distribution (mass median diameter = 0.5 μm, geometric standard deviation = 2), background ozone (80 ppb) and ammonia concentrations (10 ppb) were used, as was the MESOPUFF chemical conversion methodology.

For these 40 power plants, Wolff reported mean intake fractions of 2×10^{-7} for sulfate/SO_2 and 3×10^{-8} for nitrate/NOx. This means that for every 10 million grams of SO_2 emitted by power plants, 2 grams of sulfate are inhaled by someone in the US. This is perhaps unintuitive until we make a naïve "back of the envelope" calculation based on an earlier Abt Associates study (*5*). They modeled the benefits of power plant emission controls across the US. Their "Policy Case" resulted in a 7.1 million ton reduction in annual SO_2 emissions. 7.1 million tons per year is equal to about 2×10^{16} μg/day. We can estimate that the average ambient reduction of sulfate from this was roughly 1 μg/m^3 (looking at Exhibit 3.2 in their report). Using our above equation,

$$iF = (20\ m^3/day) * (1\ \mu g/m^3) * (290\ million\ people) / (2 \times 10^{16}\ \mu g/day) = 3 \times 10^{-7}$$

So, this simple calculation demonstrates that the magnitude of the figures is reasonable.

As discussed in Evans et al. (*6*), the nitrate/NOx intake fractions may be underestimated, as Wolff chose to divide all CALPUFF-modeled values by four to reflect known relationships between particle nitrate formation and temperature. Thus, a value of 1×10^{-7} for nitrate/NOx may be more appropriate, with the true value implied by the Wolff analysis likely falling between 3×10^{-8} and 1×10^{-7}. Because of the methodology used by CALPUFF, no nitrate/SO_2 intake fractions were estimated.

In contrast, when we apply S-R matrix to the same 40 power plants, we find mean intake fractions of 3×10^{-7} for sulfate/SO_2, 5×10^{-8} for nitrate/NOx, and -5×10^{-8} for nitrate/SO_2. In other words, S-R matrix would yield an impact due to sulfate particles approximately a factor of two higher than reported in Wolff. However, this difference is tempered somewhat by the

reduced nitrate concentrations per unit SO_2 emissions and by the higher population used by S-R matrix. When looking at NOx emissions, the S-R matrix intake fraction is between the two potential values from Wolff. Thus, there does not appear to be substantial bias in either direction, although S-R matrix yields slightly higher estimates for particle formation due to SO_2 emissions.

However, we note that the power plants in the eight electric utility systems considered in the Abt Associates report are predominantly found in the Midwest and Southeast. Because sulfate and nitrate formation patterns depend on weather patterns, it would be expected that the relationships between the models would differ across regions. Thus, our US-wide comparison for the 40 power plants may not be directly applicable to the Abt Associates analysis.

We can make a more reliable comparison for the purpose of evaluating the Abt Associates report by focusing on the seven power plants modeled in Wolff that were also modeled in the Abt Associates report. The sulfate/SO_2 and nitrate/NOx intake fractions for those seven plants are given in the table on the following page. Nitrate/SO_2 is not presented, as all values are zero in Wolff. All nitrate/NOx values are presented as reported in Wolff (*3*), with the ratios in the table reflecting both interpretations of the Wolff findings.

Plant	Sulfate/SO_2, CALPUFF	Sulfate/SO_2, S-R matrix	Ratio (S-R/CALPUFF)	Nitrate/NOx, CALPUFF	Nitrate/NOx, S-R matrix	Ratio (S-R/CALPUFF)
W H Sammis	1.6×10^{-7}	3.4×10^{-7}	2.1	2.2×10^{-8}	2.5×10^{-8}	1.1/0.3
Gorgas	1.3×10^{-7}	2.9×10^{-7}	2.2	1.6×10^{-8}	4.3×10^{-8}	2.7/0.7
Scherer	1.3×10^{-7}	3.0×10^{-7}	2.3	1.5×10^{-8}	3.9×10^{-8}	2.6/0.6
Gallatin	2.0×10^{-7}	3.5×10^{-7}	1.8	2.5×10^{-8}	4.9×10^{-8}	2.0/0.5
Cardinal	1.6×10^{-7}	3.4×10^{-7}	2.1	2.1×10^{-8}	2.5×10^{-8}	1.2/0.3
Conesville	1.7×10^{-7}	3.7×10^{-7}	2.2	2.3×10^{-8}	3.0×10^{-8}	1.1/0.3
Widows Creek	1.8×10^{-7}	3.5×10^{-7}	1.9	2.2×10^{-8}	4.6×10^{-8}	2.1/0.5

This table corroborates the general findings from the 40 plant comparison. S-R matrix yields sulfate/SO_2 intake fractions that are approximately a factor of two higher than those reported by Wolff. If we adjust the sulfate/SO_2 values for the higher population and reduced nitrate in S-R matrix, the S-R/CALPUFF ratio falls from 1.8-2.3 to 1.3-1.7. For nitrate/NOx, the two interpretations of the Wolff findings bound the S-R matrix estimates in all cases. There are some distinct geographic patterns, with values relatively lower for S-R matrix for the three plants in Ohio, versus the plants in Alabama, Georgia, and Tennessee.

Now, the critical question is: Do our findings mean that S-R matrix has overestimated sulfate formation, that Wolff's CALPUFF analysis has underestimated sulfate formation, or does the truth lie somewhere in between? Although the difference between the models is only a factor of 1.5 (a difference unlikely to lead to radically different policy decisions), understanding this question will help determine if any systematic bias exists in the Abt Associates report. Because

of the numerous differences in model assumptions, it is difficult to draw direct conclusions from the above values. The findings would be somewhat more conclusive if the two models were constructed with as many identical assumptions as possible.

In an ongoing analysis (*1*), we have modeled sulfate and nitrate impacts of seven power plants in Georgia, using both CALPUFF and S-R matrix with essentially identical model assumptions wherever possible (e.g., identical population patterns, same meteorological year, similar background pollution levels). The comparison in the following table is based on a domain within 500 km of Atlanta, making direct comparison with the values reported earlier inappropriate. Note that the nitrate/NOx intake fractions from CALPUFF do not contain the Wolff correction factor.

Plant	Sulfate/SO_2, CALPUFF	Sulfate/SO_2, S-R matrix	Ratio (S-R/CALPUFF)	Nitrate/NOx, CALPUFF	Nitrate/NOx, S-R matrix	Ratio (S-R/CALPUFF)
Bowen	1.6×10^{-7}	1.7×10^{-7}	1.1	6.7×10^{-8}	2.7×10^{-8}	0.4
Hammond	1.6×10^{-7}	1.7×10^{-7}	1.1	7.1×10^{-8}	2.7×10^{-8}	0.4
Harllee Branch	1.5×10^{-7}	1.6×10^{-7}	1.1	5.9×10^{-8}	2.3×10^{-8}	0.4
Jack McDonough	1.7×10^{-7}	1.7×10^{-7}	1.0	7.0×10^{-8}	2.6×10^{-8}	0.4
Scherer	1.5×10^{-7}	1.6×10^{-7}	1.0	5.9×10^{-8}	2.2×10^{-8}	0.4
Wansley	1.5×10^{-7}	1.8×10^{-7}	1.2	6.0×10^{-8}	2.7×10^{-8}	0.4
Yates	1.6×10^{-7}	1.8×10^{-7}	1.1	6.9×10^{-8}	2.7×10^{-8}	0.4

For sulfate/SO_2, the results are essentially identical, and are even closer when we incorporate the negative impact on nitrate formation due to SO_2 emissions in S-R matrix. This implies that the differences between S-R matrix and CALPUFF as implemented in Wolff (*3*) could be due to differences in how Wolff and Abt Associates set up their respective models rather than systematic bias related to the models themselves.

For nitrate/NOx, S-R matrix appears to systematically underestimate impacts, although we note that application of the Wolff correction factor would result in CALPUFF estimates that bound the S-R matrix estimates as above.

To understand the relative importance of these figures, we make some preliminary calculations using S-R matrix. From the above intake fraction estimates for the 40 power plants across the US, there would be about six times more exposure to particulate matter per unit emissions for SO_2 than for NOx. Since SO_2 emissions from power plants exceed emissions of NOx, the true measure of atmospheric modeling uncertainty or bias in the Abt Associates report is the uncertainty related to sulfate modeling, which is relatively insubstantial. This also has implications for our interpretation of the health evidence, as information related to sulfates will be relatively more important than information related to nitrates. In fact, my preliminary calculations using S-R matrix indicate that the sulfate impact from the power plants in the Abt Associates report actually exceeds the total particulate matter impact, due to the negative influence on nitrates.

Thus, we have shown that S-R matrix does not appear to have substantial biases in its estimation of population exposure to particulate matter. However, this does not necessarily imply that the model is correct, as it could be the case that the comparison models were biased for identical reasons as S-R matrix. Since S-R matrix was calibrated to monitored concentrations,

this provides one external checkpoint of the validity of the model. Another way that we can check the validity of S-R matrix is by examining how the non-linear patterns of sulfate and nitrate formation compare with patterns described elsewhere (*7*). Without going into great detail, the methodology used by S-R matrix to determine reactions between ammonium, sulfate, and nitrate leads to nearly identical relationships as documented by West et al. (*7*), providing further support for the validity of the Abt Associates approach.

Evaluation of Health Evidence

The second major aspect of the Abt Associates analysis we must consider is the health evidence and its validity. Since a comprehensive discussion of all health endpoints is beyond the scope of this review, I focus on selected health evidence but briefly consider three broad questions that could significantly alter the interpretation of the literature:

- Is the assumption that sulfate and nitrate particles have equal toxicity as average ambient particles valid?
- Is the assumption that ambient particulate matter levels in the model region are above any potential population threshold valid?
- Is the choice of studies for major health endpoints representative and unbiased?

Clearly, none of these questions can be resolved definitively within this document. But, the important issue is whether the assumptions made in the Abt Associates report reflect a reasonable current interpretation of the literature. In other words, it is not incumbent on Abt Associates to show definitively that all particles have identical toxicity, but rather that it is

equally likely that sulfates and nitrates are more or less toxic than average and that equality is a reasonable best estimate.

For the relative toxicity question, I paraphrase an argument presented in a recent publication (*8*). The two major cohort mortality studies (*9-12*) found significant relationships between premature mortality and sulfate concentrations, with the impact per unit concentration slightly greater than that of $PM_{2.5}$. Significant associations have also been shown in the time-series mortality literature (*13-16*). Time-series mortality studies that have not shown significant effects (*17*) have tended to have insufficient statistical power to detect effects, were they to exist. There is also limited toxicological evidence supporting sulfate health effects, although the evidence is far from conclusive. For example, some studies in rats have found respiratory effects from sulfate particles, especially in conjunction with simultaneous elemental carbon and ozone exposure (*18, 19*). Sulfur-related compounds had an effect on cardiovascular-related endpoints in dogs (*20*). There is little positive or negative evidence for nitrate particles. There is limited time-series evidence indicating positive associations between nitrate and mortality (*13, 21*), with one study that did not find statistical significance suffering from the statistical power problem cited above (*17*).

From this evidence (which represents only a brief overview of a small subset of the literature), I would conclude that it is appropriate in general to assign health impacts to sulfate and nitrate particles, although substantial uncertainties are likely. Although some evidence implies that motor vehicle-related particles could be most toxic (*22*), this study found similar toxicity for coal-related particles as for average ambient particles. In addition, the above evidence and other findings that crustal fine particles are less toxic than combustion particles (*15, 22*) are supportive of the Abt Associates assumptions. Using average particle toxicity may

underestimate or overestimate the impacts, but there is no evidence at this time strongly supportive of specific deviations in either direction. The approach taken by Abt Associates is therefore a reasonable one.

For the second point listed above, Abt Associates correctly points out that assuming a threshold would likely require one to alter the assumed slope of the concentration-response curve at concentrations above the threshold. The net effect of this on estimated benefits would be unclear. However, the literature to date has not demonstrated a threshold. Using mortality as an example, the most recent cohort study (*12*) did not show any evidence of a threshold, with annual average $PM_{2.5}$ concentrations down to approximately 9 $\mu g/m^3$. According to S-R matrix, $PM_{2.5}$ concentrations exceed this level for nearly 90% of the US population, particularly in the vicinity of the power plants modeled in the Abt Associates analysis. Furthermore, time-series studies of mortality (*23*) have found that any potential population threshold would likely be quite low. Given these points along with the fact that incorporating a threshold might either increase or decrease impacts, depending on the assumed functional form, the Abt Associates approach is reasonable and unlikely to contribute to significant bias.

Turning to the final point, I first consider premature mortality in detail, and then briefly discuss morbidity endpoints. There are two major decisions that must be made in incorporating premature mortality into a health impact analysis. The first is whether to rely on evidence from the cohort mortality literature or the time-series mortality literature, and the second is related to which studies are most representative of the selected body of literature.

It is clear on theoretical grounds that one would prefer to use evidence from cohort studies when possible, assuming that those cohort studies correctly characterize the relationship between the pollutant and the health outcome. Although studies have shown that time-series

studies with longer time windows can capture a greater magnitude of effect (*24*), a significant gap would remain provided that the effects of particulate matter are either cumulative or can extend beyond a one month period. The only logical reason to exclude cohort mortality evidence would be if one believed that the findings were spurious.

To evaluate whether this is likely to be the case and to consider the appropriate concentration-response function implied by the cohort mortality literature, we note that there are four primary cohort studies to date that provide some evidence about the effects of air pollution on mortality – the Harvard Six Cities Study (*9*), the American Cancer Society study (*10, 12*), the Adventist Health Study of Smog (*25*), and the Washington University-EPRI Veterans' Cohort Mortality Study (*26*). The first two of these studies are population-based and have undergone an extensive re-analysis (*11*). The Adventist Health Study of Smog was an analysis of residents of California who were Seventh-Day Adventists (a religious organization that largely abstains from smoking, alcohol consumption, and drug use), making it less generalizable to the population at large. The Veterans' Cohort is a study of mild-to-moderate hypertensive veterans receiving medical care for their hypertension at VA hospitals, again a population that may not generalize to the US as a whole. It had also not yet been published in final form at the time of this review. Nevertheless, we consider all four studies to some degree in the analysis of an appropriate concentration-response function.

The following table provides the core findings from the first three of these studies. The findings from the Veterans' Cohort are not included, as this study has not yet been published in final form and because the results are presented in a somewhat different format (fractional risks at mean value of pollutant less background). However, it is worth noting that this study found no

significant positive effect of $PM_{2.5}$ (and in fact, the effect was negative in some models, indicating less mortality at higher levels of $PM_{2.5}$).

The relative risks reported in the first three studies have been translated into percentage increases in mortality per $\mu g/m^3$ increase in $PM_{2.5}$ concentrations. When studies reported more than one value, I have provided a few representative values or a range. To avoid clutter in the table, I have only provided the central estimates from the models in these columns, noting when the estimates are not statistically significant, and have only given the $PM_{2.5}$ estimates. The value used by Abt Associates in their primary mortality estimate is placed in bold.

Study	Publication	% increase in mortality per $\mu g/m^3$ of $PM_{2.5}$	Notes (all table references refer to tables in original publications)
Harvard Six Cities	Dockery et al., 1993	1.2%	Using estimates from Table 3
	Krewski et al., 2000	0.8% - 1.5%	Across models in publication (Tables 3, 7, 14)
American Cancer Society	Pope et al., 1995	0.6%	Based on median PM
	Krewski et al., 2000	0.5%	Using model with mean PM rather than median (Table 31)
		0.1% - 1.2%	Across other models in publication (Tables 38, 46, 50), based on median
	Pope et al., 2002	**0.4%**	Using 1979-1983 concs.
		0.6%	Using 1999-2000 or average concs
Seventh Day Adventist	McDonnell et al., 2000	0.8%	From Table 2 (males only; not statistically significant)

A few key points emerge from this table. First, most available estimates exceed the 0.4% value used by Abt Associates. The lower values from the Krewski et al. (2000) reanalysis were in models including both $PM_{2.5}$ and SO_2. If one were to apply these values, it would be necessary to infer a causal effect of SO_2 on mortality. This inference is not well supported by the literature and has been shown to increase total health impact estimates for power plants substantially (*27*).

Thus, the estimate used by Abt Associates is a somewhat conservative value given the presupposition that a long-term exposure effect exists. If one believes that the findings for hypertensive veterans in the Washington Unversity-EPRI study represent a generalizable relationship and that the analytical methods in this study supercede the methods in the other studies cited above, then the Abt Associates estimate would not be conservative, and one would need to turn to the time-series literature for appropriate mortality estimates. I would conclude that the choice to include mortality from long-term exposure but to use a lower bound value from the literature is a reasonable decision based on currently available evidence.

Finally, I briefly consider the morbidity evidence used by Abt Associates. Broadly, the endpoints are appropriate, as they reflect respiratory and cardiovascular effects of varying ranges of severity, which is consistent biologically with available evidence. The authors took care to remove overlapping health outcomes (such as emergency room visits and hospital admissions), which is appropriate methodologically. Looking at a few specific study choices, the use of the 14-cities study (*28*) for hospital admissions for selected endpoints is appropriate, given that it employs an identical statistical methodology across all cities, minimizing the difficulty in combining evidence across studies. For many other morbidity endpoints, limited information exists in the literature, but the studies chosen by Abt Associates are representative and do not appear significantly biased. For example, for asthma attacks, a recent meta-analysis (*29*)

combined six studies to yield an estimate of a 0.3% increase in asthma attacks per $\mu g/m^3$ increase in daily PM_{10} concentrations. The study selected by Abt Associates implies a concentration-response function approximately a factor of two lower.

Conclusions

This review evaluated two critical aspects of the Abt Associates report. Through careful evaluation of the atmospheric modeling from a health-relevant perspective, we concluded that bias for sulfates was minimal. For nitrates, uncertainties appeared greater, but the small contribution of nitrates to total benefits makes this uncertainty relatively insubstantial. In terms of the health literature, the choices made by Abt Associates for mortality provided estimates at the lower end of the range in the literature provided that cohort evidence is believed. The morbidity estimates are also in line with the prevailing literature. Although significant uncertainties exist and can be quantified using information from this review and other sources, the findings from the Abt Associates report appear reasonable and useful for public policy analysis.

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The Importance of Population Susceptibility for Air Pollution Risk Assessment: A Case Study of Power Plants Near Washington, DC

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Running title: Population Susceptibility and Air Pollution Risk

Key words: asthma emergency room visits; cardiovascular hospital admissions; diabetes; education; mortality; particulate matter; power plant; risk assessment; susceptibility

Abbreviations

ACS: American Cancer Society

BACT: Best Available Control Technology

CHA: Cardiovascular hospital admissions

ERV: Emergency room visits

MMBTU: Million British Thermal Units

NIDDM: Non-insulin dependent diabetes mellitus

NOx: Nitrogen oxides

$PM_{2.5}$: Particulate matter less than 2.5 μm in aerodynamic diameter

PM_{10}: Particulate matter less than 10 μm in aerodynamic diameter

SO_2: Sulfur dioxide

Environmental Health Perspectives, in press

Abstract

In evaluating risks from air pollution, health impact assessments often focus on the magnitude of the impacts without explicitly considering the distribution of impacts across subpopulations. In this study, we construct a model to estimate the magnitude and distribution of health benefits associated with emission controls at five older power plants in the Washington, DC area. We use CALPUFF to determine the primary and secondary fine particulate matter ($PM_{2.5}$) concentration reductions associated with the hypothetical application of Best Available Control Technology to the selected power plants. We combine these concentration reductions with concentration-response functions for mortality and selected morbidity outcomes, using a conventional approach as well as considering susceptible subpopulations. Incorporating susceptibility has a minimal effect on total benefits, with central estimates of approximately 240 fewer deaths, 60 fewer cardiovascular hospital admissions (CHA), and 160 fewer pediatric asthma emergency room visits (ERV) per year. However, since individuals with lower education appear to have both higher background mortality rates and higher relative risks for air pollution-related mortality, stratifying by educational attainment implies that 51% of the mortality benefits accrue among the 25% of the population with less than high school education. Similarly, diabetics and African-Americans bear disproportionate shares of the CHA and ERV benefits, respectively. Although our ability to characterize subpopulations is constrained by the available information, our analysis demonstrates that incorporation of susceptibility information significantly affects demographic and geographic patterns of health benefits and enhances our understanding of individuals likely to benefit from emission controls.

Environmental Health Perspectives, in press

Introduction

The issue of subpopulation susceptibility to fine particulate matter ($PM_{2.5}$) has been given increased attention by researchers in recent years, motivated in part by the research priorities articulated by the National Academy of Science *(1)*. Understanding patterns of susceptibility would not only help identify and protect sensitive subpopulations, but it would also contribute to the understanding of mechanisms by which $PM_{2.5}$ might influence human health.

Often, air pollution policies are informed by risk assessments or benefit-cost analyses, which generally focus on the total health benefits of alternative emission control strategies *(2-5)*. Because limited relevant susceptibility evidence exists, differential effects on susceptible subpopulations are rarely incorporated. Typically, the same relative risks are applied to all individuals in an "at-risk" age group, and baseline disease or health care utilization rates are assumed to be uniform across large geographic areas (often national averages).

However, it is likely that the effects of air pollution vary widely across subpopulations, depending on demographics, behavior patterns, income, access to health care, and other factors. Differences could exist either in relative risks (if an increment of air pollution yields a different percentage increase in effect in different populations) or in absolute risks (if there are differences in baseline disease patterns by subpopulation, independent of air pollution). For a benefits assessment, if policy makers were concerned about distributional issues or if the ultimate valuation of benefits depended on population characteristics, the incorporation of susceptibility could potentially influence the conclusions.

One current policy issue for which information on susceptibility could be influential is the regulation of emissions from older power plants. To date, older power plants have not been required to meet the same control requirements as new sources, helping to extend the useful

lifetime of older facilities *(6-8)*. These facilities contribute a substantial fraction of national power sector emissions. In 1999, coal-fired power plants contributed approximately 86% of nitrogen oxide (NOx) emissions and 93% of sulfur dioxide (SO_2) emissions from the utility sector, largely from facilities exempted from new source standards *(9)*.

At the time this article was written, multiple states (including Massachusetts, Connecticut, and Texas) had introduced multipollutant regulations or legislation to require older power plants to meet emission levels commensurate with the application of Best Available Control Technology (BACT). Pollutants considered typically included NOx and SO_2, as well as mercury and carbon dioxide. Multipollutant power plant legislation was also being debated at the federal level, but no bills or regulations existed at the time of our analysis.

From both a state and federal perspective, the question of how the benefits of emission controls would be distributed could be important. Policy makers may be concerned about providing benefits to high-risk communities, communities near power plants, or other subpopulations. If these questions were important, population susceptibility could influence the policy choices (e.g., emission trading versus mandatory on-site controls).

In this paper, we develop a model to estimate the health benefits associated with emission reductions at older fossil-fueled power plants. We focus on both primary $PM_{2.5}$ and secondary sulfate and nitrate particles formed through emissions of SO_2 and NOx, respectively. We consider a case study of all older power plants located within a 50 mile (80 km) radius of Washington, DC. We calculate three health endpoints – premature mortality, cardiovascular hospital admissions in the elderly, and pediatric asthma emergency room visits – both using conventional assumptions and then considering available evidence for differential effects on susceptible subpopulations. Our goal is both to quantify the health benefits associated with the

implementation of BACT at the selected power plants and to consider whether introduction of susceptibility models might affect the interpretation of our findings.

Case Study Setting

For this analysis, our goal was to select a geographic area that had multiple older power plants nearby and geographic heterogeneity in factors that might influence relative risks, baseline health status, or health care utilization (such as socioeconomic status). Washington, DC and its surrounding suburbs provide an example of such a region. According to 1990 US Census data, median household income in Washington, DC ranged from under $10,000 to over $150,000 across census tracts *(10)*. Washington, DC is also quite racially divided, with few African-Americans residing in the western half of the city and mostly African-Americans residing in the eastern half of the city.

In addition, within a 50 mile (80 km) radius of Washington, there are five fossil-fueled power plants grandfathered under the Clean Air Act - Benning, Chalk Point, Dickerson, Possum Point, and Potomac River (Table 1). The choice of these five power plants is somewhat artificial, since any single regulation would not affect only these plants. However, our analysis is meant to be illustrative, and these five plants are likely the greatest contributors to heterogeneity in power plant-related exposures in the area. Inclusion of additional power plants would increase the total benefits but decrease the relative concentration gradient across the Washington area.

Methods

General

To quantify the magnitude and distribution of health benefits, we estimate the emission reductions of key pollutants, apply an atmospheric dispersion model to determine incremental concentration reductions, and derive concentration-response functions. Any such analysis involves numerous boundary decisions and contains substantial uncertainties. In this paper, we focus largely on issues related to susceptible subpopulations and the resulting implications. We do not extensively address the complexities of other elements of the model, nor do we provide a formal analysis of uncertainties. We also do not consider the economic valuation dimension of a benefits assessment. Additional information about parametric uncertainties in our atmospheric model *(4,11)* and issues related to differential particle toxicity or alternative interpretations of the health evidence *(4)* can be found elsewhere.

Quantification of emissions

We estimate emissions of $PM_{2.5}$ and its precursors (NOx and SO_2), following the model structure in our earlier analyses *(4,11)* and supported by the fact that $PM_{2.5}$ has dominated aggregate benefits in past air pollution risk assessments *(2,3)*. This omits any benefits associated with ozone, air toxics, or other impact pathways from the power sector. Of note, most proposed regulations consider NOx and SO_2 but do not directly require controls for primary $PM_{2.5}$ (although many NOx and SO_2 control strategies would affect primary $PM_{2.5}$).

We use 1999 as the base year for our analysis, evaluating the concentration and health benefits that would have been obtained had lower target emission rates been achieved. This is not

identical to the future benefits that might be obtained through pending regulation, since some facilities have ongoing or near-term plans for repowering or emission controls.

Emissions of SO_2 and NOx were taken from the US EPA Acid Rain Program Emissions Scorecard *(12)*. To capture seasonality in emissions, we incorporated quarterly average emission rates when reported. When no data on seasonal emissions were available, we assumed constant emissions per unit of heat input. For filterable $PM_{2.5}$, total plant emissions were taken from the US EPA National Emission Trends database *(13)*. We estimated condensable $PM_{2.5}$ emissions given fuel type and sulfur content, using AP-42 emission factors from US EPA.

We selected lower target emissions to correspond to the levels proposed in multiple regulations, which correspond to the application of Best Available Control Technology (BACT). This resulted in target emission rates of 0.3 lb/MMBTU of SO_2, 0.15 lb/MMBTU of NOx, and 0.01 lb/MMBTU of filterable PM. Lower target condensable PM emissions were taken from AP-42, given assumed application of control technologies. Since both Dickerson and Benning have actual filterable $PM_{2.5}$ emissions less than the lower target rate, we set the lower target filterable $PM_{2.5}$ emission rate equal to actual emissions for these plants.

Atmospheric modeling

We established a receptor grid covering a 400 km (250 mile) radius around Washington, DC (centered at 38.9°N, 77°W), to capture a significant fraction of total benefits without extending the dispersion modeling boundaries excessively (Figure 1). Because of our focus on spatial patterns, it was important to determine concentration reductions at small geographic scales close to the sources. Within 100 km of Washington, census tracts were selected, as they are relatively small (generally between 2,500 and 8,000 people) and were theoretically designed

to be socioeconomically homogeneous. Beyond 100 km, county-level resolution was used, resulting in a nested receptor grid with 1,908 receptors. Using 1990 Census data (the most recent data available at the time of our study), our receptor grid contained 47 million individuals, 7 million of whom live within 100 km of Washington.

We conducted our atmospheric modeling using CALPUFF (CALMET version 5.2 000602a, CALPUFF version 5.4-000602-1, CALPOST version 5.2 991104b; Earth Tech, Concord, MA). CALPUFF is a regional-scale Lagrangian puff model that has been recommended by US EPA for long-range transport modeling *(14)*, given that it has been shown to be relatively unbiased at distances out to 200 km *(15)*. In general, limitations in the atmospheric chemistry make the secondary pollutant estimates relatively more uncertain than the primary $PM_{2.5}$ estimates, given the nonlinearities associated with sulfate and nitrate formation.

Our methodology to generate meteorological files for CALMET was similar to the approach in our past applications and is described in depth elsewhere *(4,11)*. We combined NOAA prognostic model outputs with mesoscale data assimilation systems for each hour across our case study year (January 1999-January 2000). This involved combining lower-resolution upper air data (40 km grid spacing) generated through NOAA's Rapid Update Cycle (RUC2) model with METAR surface observations and cloud cover data available at 15 km resolution. These data sources were combined using the ARPS Data Assimilation System (ADAS) and provided hourly CALMET windfields within eight vertical layers. Precipitation data were taken from all National Climatic Data Center stations within the receptor region, with CALMET defaults used for interpolation between stations. The primary difference from our previous applications was the inclusion of 50 evenly spaced "soundings" based on columns of the ADAS

data, to more accurately provide a reasonable high-resolution temperature field and subsequent planetary boundary layer depth estimates.

In CALPUFF, we adopted recommended modeling assumptions that were used in our past applications *(4,11)*. We used the MESOPUFF II chemical transformation mechanism, which is generally preferred in urban settings. Wet and dry deposition were incorporated using precipitation data and CALPUFF default deposition rates. Hourly background ozone concentrations were taken from five CASTNET stations spaced throughout our receptor region (Prince George's, MD; Mercer, NJ; Elk, PA; Prince Edward, VA; Gilmer, WV), and we assumed a background ammonia concentration of 1 ppb.

For brevity's sake, we do not provide sensitivity or uncertainty analyses for our atmospheric modeling in this article. In our past analyses *(4,11)*, we found total benefits to be reasonably stable given single parametric changes in CALPUFF, including the chemical conversion mechanism, background ammonia concentration, and treatment of wet and dry deposition. In addition, we concluded that any bias associated with either hypothetical CALPUFF overestimation beyond 200 km or exclusion of long-range exposures is relatively small in comparison with other model uncertainties. A comprehensive risk assessment would need to incorporate these uncertainties in an evaluation of overall model uncertainty.

Health evidence

Although numerous health outcomes have been incorporated into past analyses *(2)*, we focus on a subset for which some evidence exists for differential effects on susceptible subpopulations. The choice of outcomes as well as the subpopulations considered is therefore entirely dependent on the current literature and is not meant to be comprehensive. Furthermore,

we restrict the health evidence to epidemiological studies conducted in the US, since patterns of health care utilization and the relationship between demographics and health status likely vary across countries. Given these criteria, we evaluate premature mortality (stratified by education), cardiovascular hospital admissions for the elderly (stratified by diabetic status and age), and asthma emergency room visits for children (stratified by race and age). For each outcome, we describe both a conventional approach and construct a susceptibility model. Our goal is not to consider the complete array of susceptible subpopulations, but rather to select one example for each outcome for which epidemiological evidence and population data exist.

Premature mortality

For premature mortality, we derive a central estimate from the follow-up analysis of the American Cancer Society (ACS) cohort study *(16)*. Multiple other cohort studies are available *(17,18)*, but the ACS study has the largest and most geographically diverse population, with relative risks bounded by other studies and a statistical approach suggested by a detailed reanalysis *(19)*. For all-cause mortality, the authors calculated a relative risk of 1.04 (95% CI: 1.01, 1.08) for a 10 $\mu g/m^3$ increase in annual mean $PM_{2.5}$ concentrations (using 1979-1983 concentrations). The relative risk was slightly higher (1.06) using more recent pollution data, but we use the lower figure to be conservative and since Pope and colleagues presented stratified estimates based on the 1979-1983 concentrations *(16)*.

Relative risks did not vary substantially across most demographic factors, with the exception of educational attainment. Educational attainment appeared to be a strong effect modifier across all causes of mortality. The relative risk for a 10 $\mu g/m^3$ increase in annual mean $PM_{2.5}$ concentrations was 1.085 (95% CI: 1.031, 1.142) for individuals with less than high school

education, 1.045 (95% CI: 1.004, 1.087) for individuals with high school education, and 1.003 (95% CI: 0.967, 1.040) for individuals with more than high school education.

There are numerous uncertainties related to the application of this stratified relative risk. The ACS cohort is somewhat more educated than the population at large, and correlated terms such as race and poverty status have not been significant in time-series mortality or hospital admissions studies *(20-22)*. In addition, the statistical approach implies that we are modeling the effect of education controlling for smoking and other factors, which would ideally be included to model the influence of all risk factors correlated with educational attainment. Regardless, we use the education-stratified values to determine the implications of the reported relationship.

For background mortality rates, the standard approach is to apply county-level averages to individuals age 30 and older (the age range considered in the ACS study). We use this as our baseline approach, but for our susceptibility model, consider whether mortality rates vary as a function of education while still averaging to the reported county-level rates.

There is a strong and consistent negative relationship between socioeconomic status and all-cause mortality *(23)*. Socioeconomic status can be measured by occupation, income, education, or some combination of these terms. It is generally believed that both income *(24)* and educational attainment *(25)* are independent predictors of mortality, although the bases for these relationships are not well understood. Some argue that those in lower socioeconomic classes display high-risk behaviors, such as smoking, being overweight, and not exercising *(26)*, resulting in higher mortality rates. However, only a small fraction of the increased mortality can be explained by a higher prevalence of high-risk behaviors *(27)*, so there must be other contributing factors. In any case, it is clear that those in low education or income categories represent a susceptible subpopulation for all-cause mortality.

Educational attainment is a useful predictor of mortality since it typically does not change after adulthood. Additionally, this term is available for all segments of the adult population, even those not in the workforce. Although it may be a proxy for other factors, various hypotheses have been presented for why lower education might be a causal factor for mortality. Education may be a marker for factors (such as intelligence and good health in early childhood) that allow for both educational attainment and good health in adulthood, for acquired knowledge that can be used to obtain positive health outcomes, for relative status in society, or for the development of positive social networks *(28)*. The protective effect of higher education has been seen in the US *(28)* and worldwide *(29,30)*.

We select our baseline mortality risk ratios from a study that evaluated risks for all-cause mortality as a function of both education and annual income among a cohort aged 25-64, drawn from the National Longitudinal Mortality Study *(28)*. The relationship between education and mortality was best described by a trichotomy (less than high school education, high school diploma or greater but no college diploma, or a college diploma or greater). When compared with the highest education group, the annual mortality relative risk for men was 1.7 for less than high school education and 1.5 for high school diploma or greater but no college diploma. For women, the corresponding relative risks were 1.5 and 1.2. The attenuation in women has been documented previously and can be attributed largely to the married subpopulation of women *(31)*. We apply these relative risks to all individuals over age 30, although there is some evidence that socioeconomic differences play less of a role in determining mortality rates among the aged *(32)*.

Cardiovascular hospital admissions

A number of studies in the US have evaluated the relationship between particulate matter exposure and cardiovascular hospital admissions (CHA) among individuals age 65 and older *(21,22,33-40)*. Most central estimates from these studies fall in the range of a 0.5-1% increase in CHA for a 10 $\mu g/m^3$ increase in daily PM_{10} concentrations. Using a typical $PM_{2.5}/PM_{10}$ ratio of 60%, we would consider a central estimate of an approximate 1% increase in CHA per 10 $\mu g/m^3$ increase in daily $PM_{2.5}$ concentrations appropriate. As a baseline, we apply this percentage to the average background rate of 0.084 CHA per year per individual age 65 and older *(41)*.

Although numerous factors might influence either the baseline risk or the relative risk of an air pollution-related CHA, we focus on diabetes to illustrate the influence of a risk factor that varies demographically and might influence both risks. To estimate the number of diabetic and non-diabetic CHA in a county or census tract, we consider two relationships – the risk factors for diabetes among the elderly and the differential risk for a CHA given the presence of diabetes.

In those over 65, non-insulin dependent diabetes mellitus (NIDDM) accounts for virtually all of the diabetic caseload. There are numerous risk factors for NIDDM, including age, obesity, family history, and sedentary lifestyle. Although lifestyle variables are the strongest predictors of diabetic status (accounting for as much as 90% of population attributable risk *(42)*), we cannot estimate these variables at the census tract level from publicly available data. In the absence of this information, we estimate NIDDM prevalence as a function of gender, age, and race. According to a national survey *(43)*, NIDDM prevalence in individuals over age 65 is higher among African-Americans and Mexican-Americans than in non-Hispanic whites, ranging from 10.9% for non-Hispanic white males aged 65-74 to 29% for Mexican-American females aged 65-74. We apply these estimates to our study populations, despite the limitations in applying

national relationships based on race to a specific geographic setting. The relationship between race and common risk factors likely varies widely across regions and within small geographic areas, a feature that is not captured by our model.

Regarding risks for a CHA, it has been well established that diabetics have an increased risk of heart disease. Several studies also indicate that diabetics are admitted to the hospital more frequently than non-diabetics *(44,45)*. Thus, it is unsurprising that CHA rates are elevated in diabetic populations. According to a national diabetes surveillance report *(46)*, as of 1996, the annual CHA rate was 0.20 admissions per year per diabetic age 65-74 and 0.27 for diabetics 75 and older. In contrast, the rates for the population as a whole are 0.06 (age 65-74) and 0.11 (75 and older) *(41)*. Using these two rates and the estimated diabetes prevalence across our study population, we can calculate the CHA rate for non-diabetics. Clearly, there are several appreciable assumptions underlying these estimates. Although we know that marked differences can exist in hospital utilization rates among states and communities, we assume that tract-specific rates vary only as a function of the estimated number of diabetics, with CHA rates invariant for non-diabetics. This likely underestimates the degree of spatial and demographic variability in CHA rates.

On the relative risk side, a time-series study in Chicago *(35)* found a 2% increase in CHA for diabetic individuals over age 65 for a 10 $\mu g/m^3$ increase in PM_{10}, versus a 0.9% increase for non-diabetics. In contrast, the studies that evaluated factors such as race, education, or poverty *(21,34,40)* found no significant effect modification for CHA relative risks. To ensure that our concentration-response function is in agreement with our non-stratified estimate, we assume that a factor of two difference exists between diabetics and non-diabetics and calculate the concentration-response function given the estimated number of CHA in diabetics and non-

diabetics in our study population. The result is a 0.7% increase in CHA per 10 $\mu g/m^3$ increase in $PM_{2.5}$ for non-diabetics, with a 1.5% increase for diabetics.

Pediatric asthma emergency room visits

Many studies have associated emergency room visits (ERV) for numerous respiratory and cardiovascular causes with particulate matter, but to date only two studies in the US have considered asthma-related visits among children (defined here as 18 years of age or younger). In Seattle *(47)*, an 11.6 $\mu g/m^3$ increase in PM_{10} was associated with a 14% increase in asthma ERV (95% CI: 5%, 23%), with a 9.5 $\mu g/m^3$ increase in $PM_{2.5}$ associated with a 15% increase. This study found the relative risk to be similar in high-utilization and low-utilization areas (a proxy for socioeconomic status). In Atlanta *(48)*, a 4% increase in pediatric asthma ERV was estimated for a 15 $\mu g/m^3$ increase in PM_{10} concentrations (95% CI: 0.4%, 7%). As in Seattle, there did not appear to be effect modification due to race or socioeconomic status. Simply pooling these two studies using a random effects model *(49)* provides a central estimate of a 0.7% increase in asthma ERV per $\mu g/m^3$ increase in PM_{10}, which we translate into an approximate 1% increase in asthma ERV per $\mu g/m^3$ increase in daily $PM_{2.5}$. This can be applied to a background asthma ERV rate of 0.012 for children age 0-4, 0.0081 for children age 5-14, and 0.0069 for children above age 15 *(50)*.

Although the published studies did not identify susceptible subpopulations from a relative risk perspective, the background rate of asthma ERV would be anticipated to differ widely across subpopulations. This would be a function both of trends in asthma prevalence and in patterns of health care utilization across populations.

Asthma prevalence has increased substantially in recent years *(50)*, with lower-income individuals and minorities disproportionately affected by the disease *(51-55)*. Many of the significant predictors of childhood asthma, such as cockroach presence in the home *(56)* or maternal education *(57)*, are related to socioeconomic status. Furthermore, patterns of health care utilization are strongly related to income. The ratio of anti-inflammatory to beta-agonist medication is lower in low-income communities and is inversely correlated with hospitalization rates *(58)*, and lower-income populations lacking health insurance often use emergency services as a means of primary care. Thus, it would be expected that low-income populations would have somewhat higher pediatric asthma ERV rates.

Data on pediatric asthma ERV rates as a function of income were limited, but substantial racial differences have been documented. According to data from the National Hospital Ambulatory Medical Care Survey *(50)*, across all ages, the asthma ERV rate for African-Americans is nearly five times greater than for whites (0.023 and 0.0049 per capita, respectively). No data were provided on asthma ERV rates stratified across both age and race, but a study of three-year olds in the US finds a racial differential of similar magnitude but with some independent effects of both race and income *(51)*.

Given available information, we estimate baseline pediatric asthma ERV rates as a function of age and race, assuming the racial disparity to exist in all age groups. This encompasses both differences in prevalence and in health care utilization. As with our diabetes estimates, there are some substantial limitations in using only race as a predictor, since the relationship between race and asthma ERV risk factors varies by income, urban/rural status, and other factors. Regardless, the consistent relationship between race and ERV and the ability to gather racial information at the census tract level make this the best available covariate.

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Results

Concentration Reductions

Using our atmospheric dispersion model, the emission reductions at the five selected power plants lead to annual average $PM_{2.5}$ (primary plus secondary) concentration reductions ranging from 0.009-0.9 $\mu g/m^3$ in our receptor region (Figure 2). By way of comparison, according to EPA AIRS data, annual average $PM_{2.5}$ concentrations in Washington were approximately 14-18 $\mu g/m^3$ in 1999. The maximum annual average $PM_{2.5}$ concentration reduction is found within Washington, as might be anticipated by the power plant selection criteria and the inclusion of primary $PM_{2.5}$.

The geographic distribution of benefits varies somewhat across particle types, power plants, and seasons. Annual average primary $PM_{2.5}$ concentration reductions peak closer to the plants and decrease more rapidly with distance than secondary sulfates or nitrates (Figure 2). As a result, a greater fraction of total exposure reduction (defined as the sum across receptors of the product of concentration reduction and population assigned to the receptor) occurs closer to the power plants for primary than for secondary $PM_{2.5}$ (Figure 3). However, there is tremendous variability in the distribution of total exposure reduction, principally due to variations in source locations and pollutant type (primary versus secondary). In addition, total exposure reduction per unit emissions displayed expected seasonal patterns, with slightly higher values for primary $PM_{2.5}$ in the winter and fall (related in part to lower mixing heights) and higher values for sulfates and lower values for nitrates in the summer due to the effect of temperature on relative conversion rates.

Health Benefits

For premature mortality, using non-stratified relative risks and homogeneous baseline mortality rates within counties, our central estimate is that emission reductions from the five power plants would lead to 210 fewer deaths per year (Table 2). The estimated impact under the current emissions scenario is 270 deaths per year. Of the total mortality benefits, approximately 25% occur in individuals with less than high school education (identical to the proportion in the population). Approximately 16% of mortality benefits accrue within 50 km of the power plants, largely related to the substantial contribution of secondary sulfates (62%) and nitrates (19%) to total $PM_{2.5}$ exposures.

In our susceptibility model, with both baseline mortality rates and $PM_{2.5}$ relative risks stratified by educational attainment, our understanding of the affected subpopulations changes substantially (Table 2). The total mortality benefit is largely unaffected, with a slight increase associated with differences in educational attainment between the Washington area and the ACS cohort. However, 51% of the estimated mortality benefits now accrue among individuals with less than high school education, double the prediction in the homogenous risk model.

Although stratification by education does not significantly influence the broad geographic patterns of benefits (i.e., the fraction of benefits within 50 km), at the census tract level, benefits differ by as much as a factor of 13 between the models. Figure 4 depicts the geographic patterns of benefits under both the baseline and susceptibility models, focusing solely on census tracts in Washington, DC for simplicity. Using the baseline model, the mortality risk reductions in Washington are reasonably homogeneous, ranging from 36 to 67 fewer deaths per year per million individuals over age 30. Under the education-stratified model, the range broadens considerably and the distribution is more complex, with per capita benefits now ranging by more

than a factor of 10 across census tracts. The mortality benefits are generally increased in southeastern Washington, the lowest-income area of the city.

When we consider CHA among the elderly, our baseline model estimates 59 fewer CHA per year. Although it seems counterintuitive that the mortality numbers could exceed the morbidity numbers, this is related to the limited focus on cardiovascular admissions due to only short-term exposures among the elderly (versus all-cause mortality from long-term exposures among individuals age 30 and older). Using a conventional model that assumes diabetics not to differ in any way from non-diabetics, 13% of the CHA benefits are estimated to occur among diabetics, while 80% are found among non-Hispanic whites (Table 2). The geographic distribution of CHA benefits is similar to the exposure reduction and mortality benefits, with differences reflecting the relative number of individuals age 65-74 and above age 75 within census tracts.

As expected, incorporating the diabetes-based information has a minimal impact on aggregate benefits but dramatically alters the profile of the affected individuals (Table 2). Using this model, 54% of the CHA benefits are found among diabetics, with 76% among non-Hispanic whites. Since we have assumed that baseline CHA risk for non-diabetics does not differ as a function of race or income, the CHA estimates under the susceptibility model are closer to those from the baseline model than for mortality (Figure 4). However, even only considering diabetes-related susceptibility changes the census tract-level benefits by as much as 40%.

Finally, we estimate 140 fewer pediatric asthma ERV per year using our non-stratified model (38% in children age 0-4, with 46% in children age 5-14). Twenty-seven percent of benefits occur in African-American children (who represent 21% of the study population). When we stratify asthma ERV risk by race, the total benefits increase to 160 fewer visits per year, with

significant changes in the geographic and demographic distributions (Table 2). The census tract-level risk reduction varies by an order of magnitude across Washington, with the benefits increased by more than a factor of two in the eastern half of the city (Figure 4). The proportion of benefits among African-American children is increased to 64%, commensurate with the assumption of greater baseline asthma ERV rates.

Discussion

Our analytical approach demonstrates two important points. First, given an interpretation of the epidemiological evidence that assumes that ambient concentrations in the Washington, DC area exceed any potential population threshold for $PM_{2.5}$ health effects, emission controls at older fossil-fueled power plants would provide tangible and quantifiable health benefits. Second, when we take account of susceptible subpopulations and differences in both relative risk and baseline disease rates across these populations, the small-scale geographic and demographic distributions of those benefits are strongly affected. For the example of premature mortality, if educational attainment influences both the relative risk of air pollution and the baseline mortality risk, then more than half of the mortality benefits accrue among the 25% of our study population with less than high school education. Similarly, for pediatric asthma emergency room visits, the fact that background rates are substantially greater in African-Americans implies that a majority of the emergency room visit benefits accrue in 21% of the population, even given identical relative risks from air pollution. The relatively smaller differences found for cardiovascular hospital admissions when diabetes is considered illustrates that evidence for differential effects on a relatively small fraction of the population has a smaller effect than a population-wide model.

There are clearly some barriers in both interpretation of the study findings and application of our model to other settings. One important uncertainty is related to the stratified risk models we selected. For all health outcomes, we used stratification variables (such as race) that might have independent effects on baseline health but likely are proxies for numerous socioeconomic endpoints. If the stratification variables represent other factors, this adds to the uncertainty in a site-specific stratified analysis.

In general, we have applied susceptibility models based on national data to a small number of states, which has multiple inherent limitations. Clearly, it would be preferable to use local health data, but data at small geographic scales for a large region are difficult to obtain and are rarely stratified across all demographic variables of interest. In addition, the reliance on national data increases the generalizability of our findings. Despite these issues, our models demonstrate that simple assumptions about susceptibility can be influential in our understanding of health risks and benefits. The alternative is an assumption of homogeneity, which itself introduces implicit uncertainty and may contribute to biases in selected settings.

Another limitation of our study is the fact that we have devoted limited attention to uncertainty analysis, a crucial element in interpreting sensitive and complex findings. Drawing on the uncertainty analyses in our earlier work *(4,11)*, most parametric changes in CALPUFF led to changes to aggregate benefits of less than a factor of two, while variations in concentration-response assumptions (particularly for mortality) could influence estimates by as much as a factor of five. The influence of population susceptibility is generally at the lower end of this range, even for small geographic scales. However, susceptibility information has a greater influence on the relative distribution of benefits than other assumptions, many of which tend to affect all populations identically (e.g., the population average concentration-response).

Furthermore, a broader view of areas of heterogeneity or susceptibility (e.g., assumptions regarding particle size and chemical composition, time-activity data, or physiological factors *(59)*) could increase the importance of this evidence. Further analysis that considered the full array of uncertainties and evaluated which (if any) would be influential in policy decisions would be warranted.

In addition, although we have focused on power plants (in part due to pending regulatory decisions at the time of our analysis), the issue of susceptible subpopulations is likely more significant for motor vehicle pollution. Given that motor vehicles have low stack heights and have a strong presence in urban street canyons with high population density, it is likely that aggregate impacts would be spread over a smaller population than for power plants. If the exposed population had demographic differences from the US average, assumptions of homogeneity would bias the risk calculations.

Finally, any assessment of impacts from a limited number of sources is somewhat impaired by the relatively small reductions when compared with baseline concentrations. This makes field validation of model results difficult and implies that an ultimate comparison of the costs and benefits of taking action would be required to determine if action is warranted.

Despite these limitations, our analysis illustrates that emission controls at older fossil-fueled power plants could lead to quantifiable concentration and health benefits and that susceptibility information informs the interpretation of those benefits. Although the individual benefits represent a small increment over baseline risks, the number of people affected due to long-range pollution transport implies aggregate benefits that are relevant for policy evaluation. As the health literature develops additional information about differences in relative and absolute

risk across populations, risk assessments and benefit-cost analyses should take advantage of this information to provide more interpretable information to decision makers.

Conclusions

We have evaluated the health benefits of emission controls at five older fossil-fueled power plants in the Washington, DC area, using both conventional risk assessment assumptions and incorporating available information about susceptible subpopulations. We find that the geographic and demographic distribution of benefits differs substantially between the two approaches. If robust and causal, our susceptibility models identify subpopulations that bear a disproportionate air pollution burden and account for a substantial fraction of the benefits of emission controls (lower-educated individuals for mortality, diabetics for cardiovascular hospital admissions, and African-Americans for asthma emergency room visits). The characterization of high-risk subpopulations can help both in the interpretation of the risk assessment and in targeting future exposure assessment or epidemiological efforts.

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Table 1: Characteristics of five power plants in Washington, DC case study (1999 data).

	Benning	Chalk Point	Dickerson	Possum Point	Potomac River
Initial year of commercial operation	1968	1964	1959	1948	1949
Nameplate capacity (MW)	580	2046	588	1373	514
Heat input (MMBTU)	3,304,107	85,352,274	33,592,811	28,930,805	32,100,184
Emissions (Tons, % per quarter)					
SO_2	1,432 (2,21,76,2)	57,630 (21,25,31,23)	30,637 (30,17,34,18)	19,497 (24,22,32,23)	17,627 (22,28,29,21)
NOx	447 (2,22,74,1)	25,222 (20,24,30,26)	10,709 (30,17,34,18)	5,116 (25,22,32,21)	6,893 (21,28,30,21)
$PM_{2.5}$	12 (2,22,74,2)	304 (21,27,33,20)	14 (30,17,34,18)	156 (23,20,37,20)	106 (21,28,29,22)

Table 2: Magnitude and distribution of health benefits associated with modeled emission reductions at five power plants near Washington, DC (rounded to two significant figures; sums may not add due to rounding).

	Baseline model (No stratification)	Full susceptibility model (Stratification by listed covariate)
Deaths/year		
Total	210	240
< HS education	52	120
≥ HS education	150	120
Cardiovascular hospital admissions/year		
Total	59	60
Diabetic	8	33
Non-diabetic	51	27
Asthma emergency room visits/year		
Total	140	160
African-American	38	100
Non-African-American	100	57

Figure 1: Receptor grid and power plant locations for Washington, DC case study.

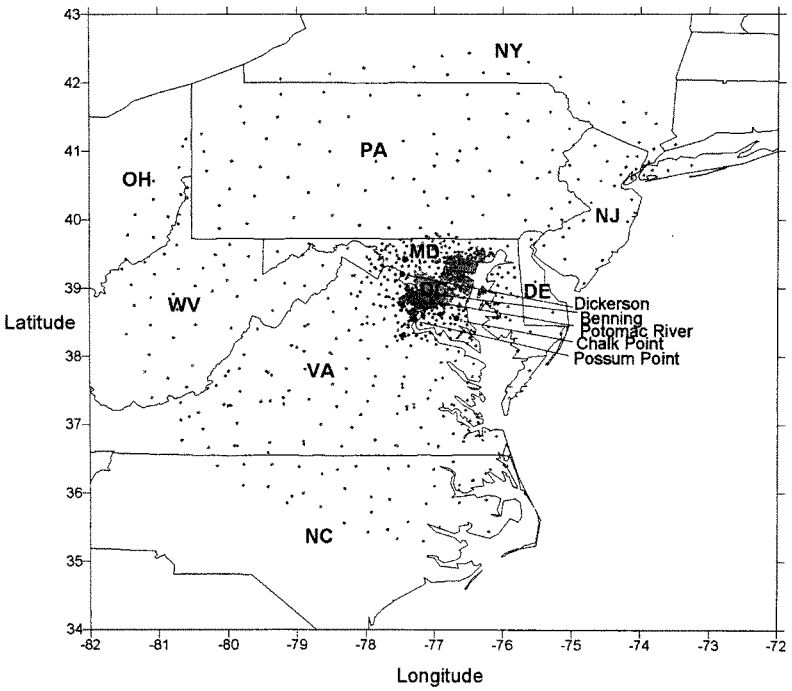

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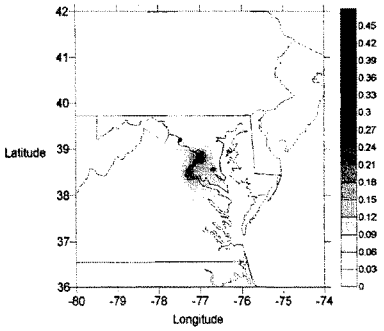
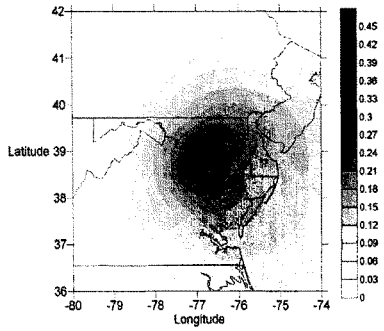
Figure 2: Combined concentration reductions (annual average, $\mu g/m^3$) from hypothetical emission controls at five power plants (primary $PM_{2.5}$, secondary $PM_{2.5}$, and total $PM_{2.5}$).

a) Primary $PM_{2.5}$



b) Secondary $PM_{2.5}$



c) Total $PM_{2.5}$

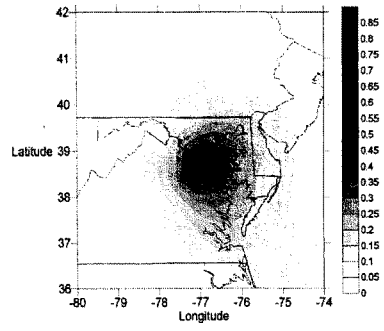


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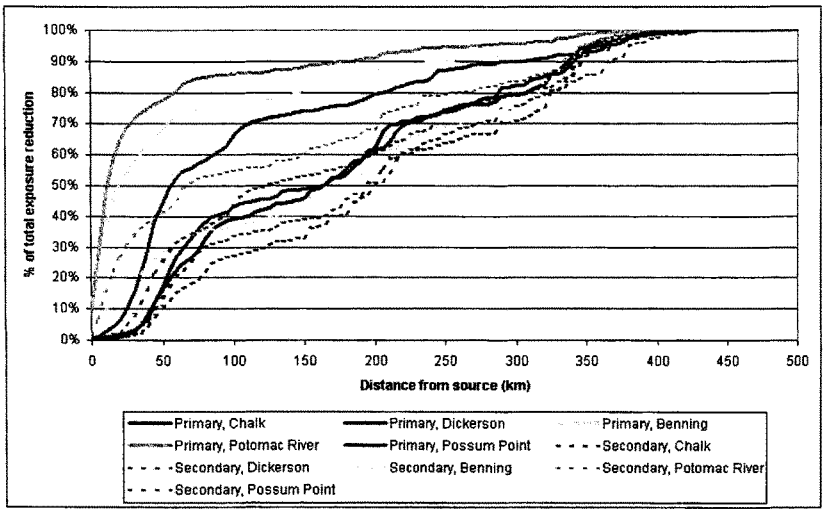
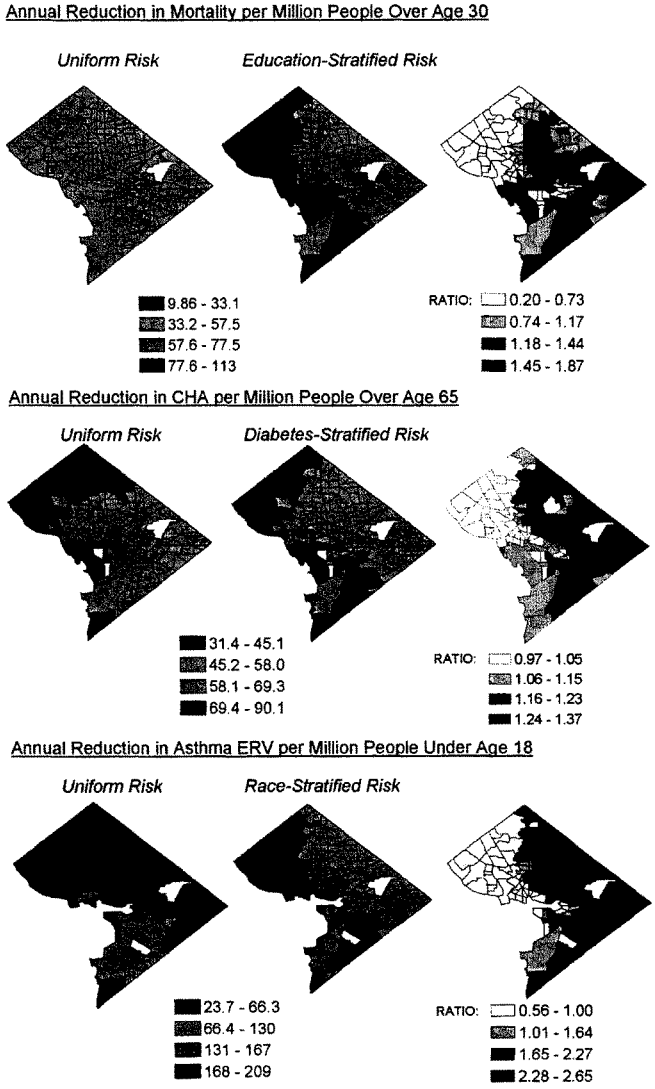
Figure 3: Cumulative distribution of total exposure reduction as a function of distance from the source, by power plant and pollutant type.



Environmental Health Perspectives, in press

Figure 4: Distribution of health benefits by census tract in Washington, DC (no color indicates zero at-risk population).



TECHNICAL PAPER

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Modeling the Benefits of Power Plant Emission Controls in Massachusetts

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ABSTRACT

Older fossil-fueled power plants provide a significant portion of emissions of criteria air pollutants in the United States, in part because these facilities are not required to meet the same emission standards as new sources under the Clean Air Act. Pending regulations for older power plants need information about any potential public health benefits of emission reductions, which can be estimated by combining emissions information, dispersion modeling, and epidemiologic evidence. In this article, we develop an analytical modeling framework that can evaluate health benefits of emission controls, and we apply our model to two power plants in Massachusetts. Using the CALPUFF atmospheric dispersion model, we estimate that use of Best Available Control Technology (BACT) for NO_x and SO_2 would lead to maximum annual average secondary particulate matter (PM) concentration reductions of 0.2 $\mu g/m^3$. When we combine concentration reductions with current health evidence, our central estimate is that the secondary PM reductions from these two power plants would avert 70 deaths per year in a population of 33 million individuals. Although benefit estimates could differ substantially with different interpretations of the health literature, parametric perturbations within CALPUFF and other simple model changes have relatively small impacts from an aggregate risk perspective. While further analysis would be required to reduce uncertainties and expand on our analytical model, our framework can help decision-makers evaluate the magnitude and distribution of benefits under different control scenarios.

IMPLICATIONS

Older power plants are not required to meet the same emission standards as new facilities and contribute a substantial fraction of criteria pollutant emissions in the United States. We have developed a model to evaluate the health benefits associated with NO_x and SO_2 emission reductions. Our central estimate is that requiring two older coal-fired power plants in Massachusetts to use BACT would lead to ~70 fewer premature deaths per year due to reduced secondary PM exposure. Along with detailed information about uncertainties and costs, benefit estimates can be used to help guide policy for older power plants.

INTRODUCTION

Because of a "grandfathering" provision in the Clean Air Act, older power plants have not been required to meet the same control requirements as new sources. This has created economic incentives to continue the usage of older facilities and discouraged new entrants in the power sector.[1-3] As a result, a small number of older power plants are responsible for a significant fraction of national emissions of SO_2 and NO_x. According to U.S. Environmental Protection Agency (EPA) data, the power sector is responsible for ~67% of national SO_2 emissions and 28% of national NO_x emissions, of which pre-1980 coal-fired power plants are responsible for 97 and 85%, respectively.[4]

To remedy this situation, some states have proposed multipollutant regulations to require grandfathered power plants to meet Best Available Control Technology (BACT) requirements, with national legislation also introduced. The state-level debates regarding the proposed regulations have implicitly and explicitly focused on two major questions. First, will the potential benefits of emission reductions (e.g., human health, ecosystem health, climate change, leveling the economic playing field) justify the potential costs (e.g., increased electricity prices, reduced fuel diversity, decreased system reliability)? Second, should emissions limits be met by mandatory on-site reductions, through a national emissions trading system, or through a combination? In contrast, there has been only moderate debate about whether removing the grandfathering provision would lead to fewer violations of National Ambient Air Quality Standards (NAAQS) in the short-term or the long-term.

This focus can be related to two key issues. First, there has been growing emphasis on the need for a cost-effective means of improving ambient air quality. Second, and perhaps more critically, the scientific literature for many criteria pollutants and health effects has been unable to detect the existence of population thresholds. States that are above and below the NAAQS are evaluating these

control strategies in a similar framework. This implies that a risk-based "damage function" approach may be the most reasonable way to understand the benefits of control policies while addressing the critical questions listed above.

Adopting a risk-based framework for policy formulation is sensible on numerous fronts. For example, under a framework where the primary focus is on control of air pollution to avoid exceeding thresholds, sources with tall stacks that have more disperse concentration impacts but significant effects on aggregate exposure may be overlooked. Moving to a risk-based framework of analysis can allow for better prioritization of control from a public health standpoint, while incorporating concepts such as cost-effectiveness and environmental equity.

Multiple studies in recent years have used this framework to analyze the impacts associated with emissions from power plants[5-8] or from transportation sources.[9,10] All of these studies ultimately attributed a health and subsequent economic burden to the incremental emissions from specific sources or the equivalent calculation of the benefits of emission reductions from these sources. Lifecycle impact assessments can be viewed in a similar light, as they aim to estimate the range of impacts associated with a specific product or process. Although these studies and similar externality assessments have been conducted for decades, they have had limited regulatory application.

Although the methodologies differ widely across impact assessment types and practitioners, all of these studies follow a general framework that merits careful scrutiny. For any damage/benefit estimation, the primary components can be categorized generally as

- estimation/forecasting of the amount of goods produced,
- evaluation of the required inputs to produce these goods,
- emissions inventory/estimation,
- dispersion modeling/exposure assessment,
- estimation of impacts associated with pollutant exposures among at-risk subpopulations or systems, and
- valuation and aggregation of impacts.

A comprehensive evaluation of the benefits of controls requires each of these components to be modeled with some precision and with adequate characterization of uncertainty, and addressing all components in depth is beyond the scope of this paper. In this paper, we propose a model framework to quantify the human health benefits associated with emission reductions at fossil-fueled power plants, with limited quantitative and qualitative consideration of uncertainty in critical components. Because of the numerous elements embedded within a damage function model, we attempt to list the uncertainties and assumptions we consider critical for accurate benefit estimation for power plants (generalizable in part to other source categories). For each component listed above, we consider the uncertainties that might significantly affect model results. To demonstrate the implications of varying assumptions in a subset of model components and to quantify the general magnitude of benefits associated with controlling emissions from grandfathered power plants, we present the findings from a case study in Massachusetts.

ANALYTICAL MODEL

Case Study Framework

To help frame the presentation of our analytical model, we first provide some details about our case study. We focus on two power plants in Massachusetts—Brayton Point (Somerset) and Salem Harbor (Salem). Massachusetts was selected because the state and stakeholders were debating regulation of grandfathered power plants at the time of our analysis. In addition, the Brayton Point and Salem Harbor plants are two of the higher-emitting facilities among the six grandfathered power plants affected by pending regulation. In total, these six power plants contribute approximately half of total SO_2 emissions and point source NO_x emissions in Massachusetts (8% of total NO_x emissions).[11] The Brayton Point and Salem Harbor power plants provide more than half of the grandfathered power plant contribution.[12] Both power plants are largely coal-fired, and basic characteristics of both facilities are provided in Table 1.

The regulation finalized in Massachusetts in April 2001 (310 CMR 7.29) contained emission standards for NO_x, SO_2, Hg, and CO_2, along with a placeholder to add primary fine particulate matter ($PM_{2.5}$) control at a later date. To parallel this regulation, we focus primarily on the potential health benefits associated with NO_x and SO_2 controls, through reductions in secondary PM concentrations. This omits benefits associated with Hg and CO_2, as well as any benefits associated with reductions

Table 1. Characteristics of power plants for Massachusetts case study.

	Salem Harbor	Brayton Point
Initial Year of Commercial Operation	1952	1963
Nameplate Capacity (MW, 1998)	805	1611
Net Generation (MWh, average 1996–1998)	3,222,262	7,660,738
Heat Input (MMBTU, average 1996–1998)	44,139,484	84,210,445
Emissions (Tons, average 1996–1998)		
SO_2	30,100	46,500
NO_x	6300	14,400

in primary PM (filterable and condensable), ozone, gaseous pollutants, or air toxics. This implies that our primary benefit estimates should not be taken as total benefits of controls but rather as a subset of benefits anticipated to contribute significantly to the total.

To evaluate benefits in the context of this state regulation, we consider two emission scenarios: one representing current practice, and one representing lower target emissions achievable through the application of BACT. Actual emissions of SO_2 and NO_x were estimated as the 3-year average of emission rates between 1996 and 1998 (the most recent available data at the time of our analysis). For lower target emissions, emission rates under BACT for coal-fired power plants built in recent years were 0.30 lb/MMBTU for SO_2 and 0.15 lb/MMBTU for NO_x. These values closely parallel the target values in Massachusetts of 3 lb/MWh for SO_2 and 1.5 lb/MWh for NO_x. Finally, for this case study, we focus exclusively on premature mortality. Although multiple morbidity outcomes have been linked with $PM_{2.5}$ exposure and could have significant contributions to monetized benefits under some valuation approaches, premature mortality has been the largest contributor in past studies.[5-10,13,14]

Damage Function Framework

As mentioned above, numerous subanalyses are contained in models to estimate the benefits of source controls, and the precise elements in these subanalyses clearly depend on the project framework. In Table 2, we list the major components of a damage function model for estimating the human health impacts of power plants, including a comparison between the idealized model and the assumptions made for our case study. It should be noted that the focus on air emissions and human health is a boundary decision underlying this figure that omits multiple categories of pollutants and effects. In addition, we provide our qualitative assessment of the direction and potential magnitude of any biases (small, medium, or large) associated with our case study assumptions. The ranking of the magnitude of biases is entirely subjective and at this time cannot be translated into quantitative uncertainty bounds. In general, "large" uncertainties are those that might potentially invalidate the findings or alter estimates by as much as an order of magnitude, while "small" uncertainties would be unlikely to influence any policy decisions. In the following section, we briefly describe the major analytical issues and uncertainties for each component, focusing on the choices made in our case study and their possible implications. We focus most extensively on the health evidence and what can be assumed about the benefits of incremental decreases in $PM_{2.5}$ concentrations at current ambient levels.

Production Forecasting

A comprehensive assessment of benefits would need to evaluate the path of electricity production across time for the affected power plants, both under current regulatory and consumption trends and given the additional regulation. For a state-level regulation (the focus of our analysis), there are two plausible extreme scenarios. In the first case, the regulation would not greatly influence utilization patterns or encourage new entrants in the marketplace, while growing electricity demand over time would result in increased utilization at previously grandfathered power plants. This would limit the benefits of controls. At the other extreme, any required installation of control technology could increase costs and lead to reduced utilization, which would increase the benefits of requiring plants to meet the lower emission levels (provided that the replacement electricity had lower impacts per unit generation). Some of the critical uncertainties for production forecasting are related to the structure of the regulation; a regulation mandating on-site clean up would likely have different implications than a regulation with an emissions trading framework. Regardless, accurate long-term modeling of benefits requires the application of economic and energy consumption models to estimate individual plant utilization.

In our case study, we assume that all units at the facilities precisely meet the lower target levels achievable through the application of BACT, with no changes in utilization. It is likely that changes in the electricity market in the northeast coupled with pending regulations would have some influence on individual plant utilization, but plant-specific projections are unavailable, and no specific deviation from constant utilization is warranted. Given that utilization could move in either direction, this provides a reasonable central estimate of benefits, particularly in the near-term when substantial market changes are unlikely. This element of uncertainty is not incorporated into our quantitative analysis.

Input Estimation/Emissions Inventory

The emissions per unit electricity generation can potentially be the most straightforward component of the damage function model, if we do not adopt a life-cycle approach and if emissions of key pollutants have been directly measured during the relevant time period. In our case study, we have accurate estimates of both past emissions and future target emissions of SO_2 and NO_x. Substantial uncertainties can be related to the determination of system boundaries. If the goal of the analysis is only to estimate impacts from stack emissions, this problem is less crucial. However, even an impact assessment of stack emissions may omit important impacts by focusing on a limited number

Table 2. Components of a damage function model for health effects of air emissions from power plants.

Component	Description	Case Study Assumption	Likely Direction/ Magnitude of Bias from Assumption
Production forecasting	Evaluation of electricity production over time, under current regulatory/consumption trends, and given additional regulation	Current utilization, focus on near-term	?; unknown
Emissions inventory	Estimation of time-resolved emissions of key pollutants per unit of electricity generation under multiple scenarios, potentially including upstream sources	Only stack emissions of NO_x and SO_2 (primary PM, air toxics omitted)	↓; medium
Atmospheric modeling	Calculation of annual average concentration increment associated with power plants across receptor region	600 × 600 km modeling regime	↓; small
		Use of MESOPUFF II, default wet/dry deposition, default NH_3 concentrations	?; medium
		Use of CALMET/CALPUFF	?; unknown
		Concentrations correlated with exposures	?; small
Health effects	Determination of morbidity and mortality effects associated with modeled concentration increments	Estimation of only mortality	↓; medium
		Assumption that ACS represents correct $PM_{2.5}$–mortality relationship	?; large
		Equal toxicity of all particles	?; large
		Linear concentration-response with no threshold	↑; unknown
At-risk population	Within each census tract, determination of the population to evaluate for health impacts and the baseline rate of disease/death	Identical relative risk for all people >30, identical background mortality rates in all census tracts	?; medium
Valuation	For each health outcome, assign an economic or health-based value corresponding to the outcome for the relevant at-risk population	Not addressed; EPA VSL estimate used for illustrative purposes	↑; large

of pollutants or by omitting other exposure pathways (e.g., fly ash). We address this in part in our case study by incorporating primary PM (filterable and condensable) in our sensitivity analysis.

In addition, uncertainties can be found when the emissions inventory does not provide the information needed for accurate dispersion modeling, such as particle size distributions or time-resolved emissions. To address the potential importance of the latter point, we evaluate benefits assuming both uniform emissions across the year and using seasonally varying emissions. Because of seasonal patterns in electricity demand and planned outages, there will clearly be some variation in emissions (particularly if we were to model peaking rather than base load units). Often, only annual average tonnage data are available, so it is important to determine if the assumption of uniform emissions might lead to significant errors.

Atmospheric Modeling/Exposure Assessment

There are numerous uncertainties and methodological issues in evaluating exposures per unit emissions, including the relevant exposure period, the geographic area of concern, the estimation of meteorological patterns, the determination of chemical conversion and deposition, and the relationship between ambient concentrations and personal exposures. In this article, we briefly discuss the choices made for our case study and the potential implications. More detailed information about our atmospheric modeling choices and the sensitivity of model findings to these choices in a different geographic setting can be found elsewhere.[15]

As outlined in a subsequent section, cohort studies provide an association between premature mortality and annual average $PM_{2.5}$ concentrations, so we focus our atmospheric modeling on estimating annual average $PM_{2.5}$ reductions at each receptor point. Of note, assuming a linear concentration-response function at current ambient levels implies that this exposure measure would also be applicable for time-series studies (because the average of the daily impacts would equal the daily impacts of the average). We construct our dispersion model to evaluate a geographic region covering 40–45° N and 67–75° W (~600 km × 600 km), with receptors at each census tract (see Figure 1). In total, this results in a potentially affected population of ~33 million, including 6 million in Massachusetts and 13 million in New York. This modeling domain was selected to maintain reasonable accuracy for the dispersion model while incorporating a significant fraction of aggregate impacts. Because of the importance of long-range transport of secondary pollutants, our receptor region likely omits a portion of total impacts, and the magnitude of this omission is estimated in our sensitivity analysis.

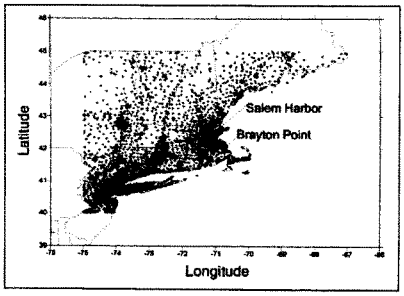

Figure 1. Receptor region for atmospheric dispersion model.

For the atmospheric modeling, we selected the CALMET/CALPUFF modeling framework.[16] CALPUFF is a Lagrangian puff model that can handle complex 3-dimensional windfields and has been shown to be unbiased for distances up to 200 km (with potential application at much longer range).[17] EPA has recommended CALPUFF for use in long-range transport modeling,[18] with the caveat that secondary particulate formation contains some uncertainties due to the first-order chemistry in CALPUFF and the complex nonlinearities in the sulfate-nitrate-ammonia-water system. We chose CALPUFF over other regional-scale models (e.g., UAM, Models-3, REMSAD) because of the relative ease of running the model for single sources under numerous parametric assumptions. Our modeling methodology using CALMET/CALPUFF is described at length in a separate publication.[15]

Briefly, we developed CALMET meteorological data by combining National Oceanic and Atmospheric Administration (NOAA) prognostic model outputs with meso-scale data assimilation systems for each hour across 1 year (January 1999–January 2000). NOAA's Rapid Update Cycle model provided upper air data at 40-km grid spacing. To provide the greater resolution needed to capture ground-level features, the Advanced Regional Prediction System (ARPS) Data Assimilation System was used in conjunction with METAR surface observations and reported cloud cover. The resulting hourly CALMET windfields had 15-km spacing within eight vertical layers. Precipitation data were taken from all National Climatic Data Center stations within the receptor region, with CALMET defaults used for interpolation between stations.

Within CALPUFF, we applied the MESOPUFF II chemical transformation mechanism and estimated wet and dry deposition using CALPUFF default parameters. We used hourly background ozone concentrations taken from CASTNET stations within the region (Woodstock, NH;

Connecticut Hill, NY; Washington Crossing, NJ). We assumed a constant NH_3 concentration of 1 ppb, which is an order of magnitude less than the CALPUFF default of 10 ppb and was selected to reflect the lower NH_3 levels in the northeast (to avoid overestimating nitrate formation). In this article, we address parametric uncertainty through the application of alternative assumptions for chemical conversion mechanism, implementation of wet and dry deposition, and assumed background NH_3 concentrations. However, it is difficult to evaluate whether the CALPUFF model is unbiased when compared with other atmospheric dispersion models, indicating that there remains significant model uncertainty that can only be evaluated indirectly in our analysis.

Finally, the issue of the relationship between concentration and exposure is not addressed in our analysis. Epidemiologic studies evaluate the relationship between health outcomes and ambient concentrations as recorded by central-site monitors, which correspond directly with the results from our dispersion models. These results are interpretable provided that ambient concentrations are correlated with population mean personal exposures to outdoor-generated pollutants. Because fine particles penetrate into the indoor environment with extremely high efficiency (particularly in well-ventilated settings),[19] this interpretation is likely appropriate. The critical question is whether any bias is induced due to differences between concentration and exposure. It has been argued that the difference between monitored and true ambient levels and the individual's deviation from risk-weighted average personal exposure would be Berksonian errors (which would not bias concentration-response functions), while the difference between average personal exposure and ambient levels could induce bias (most likely underestimating the effect).[20]

Concentration-Response Estimation

The key issue in determining appropriate concentration-response functions is to estimate the slope of the curve at current ambient concentrations. It should be noted that it is not necessary to determine whether a population threshold exists but, rather, whether there is evidence of a threshold above current ambient concentrations. In addition, because concentrations below NAAQS levels do not necessarily correspond to a zero risk level,[21] we must look to the health literature to determine if effects appear to be present at current ambient concentrations. In evaluating the health literature, we also must attempt to allocate effects to only the pollutants causally associated with the health outcomes. While misallocation could have limited influence on baseline risk calculations, it would have significant implications for the benefits of pollutant-specific control strategies. In this article, we focus on premature mortality associated with $PM_{2.5}$ exposure, motivated by its relative importance in past benefit-cost analyses.[13,14] In this section, we attempt to derive a reasonable central estimate of mortality impacts, and we discuss the substantial uncertainties in the Sensitivity Analysis section.

We consider the cohort mortality literature to capture any potential long-term exposure effects. To date, there have been three major published cohort mortality studies—the American Cancer Society study (ACS),[22] the Six Cities study (SC),[23] and the Adventist Health Study of Smog (AHSMOG).[24] The first two of these were recently reanalyzed by the Health Effects Institute (HEI).[25] In addition, preliminary findings from a national prospective cohort study of male veterans (VA) have been reported.[26] In general, the $PM_{2.5}$ effect is greatest in SC and smaller (and nonsignificant) in AHSMOG and VA. Because the ACS estimates are generally bounded by other studies, we derive our central estimate from ACS. In addition, more substantial concerns have been voiced about the other studies, including sample size and inability to discriminate among pollutants in SC, sample size and representativeness and lack of measured $PM_{2.5}$ in AHSMOG, and representativeness and inclusion of excessive covariates in VA.[27] Nevertheless, differences in findings among the studies should be included in a comprehensive uncertainty analysis and are considered to a limited extent in our sensitivity analysis.

The ACS study was a retrospective analysis of a cohort of more than 500,000 adults across the United States, followed from 1982 to 1989.[22] The cohort consisted of individuals at least 30 years of age at the time of enrollment who were generally acquaintances of volunteers for ACS (raising the question of population representativeness). To estimate air pollution exposures, individuals were matched to the nearest ambient monitors using concentrations from the start of the study period. In the original publication, the authors reported a relative risk of 1.17 for a 24.5 $\mu g/m^3$ increase in annual median $PM_{2.5}$ concentrations (95% CI: 1.09, 1.26), with a relative risk of 1.15 for a 19.9 $\mu g/m^3$ increase in annual mean SO_4^{2-} concentrations (95% CI: 1.09, 1.22).

In the HEI reanalysis,[25] numerous statistical models were tested to evaluate the robustness of this finding, with $PM_{2.5}$ relative risk ranging from insignificant to double the original estimate. Our central estimate is derived from a model using mean $PM_{2.5}$ concentrations from dichotomous samplers and including individual-level covariates for tobacco consumption, education, occupational exposure, body mass index, marital status, and alcohol consumption. Using this model, the authors calculated a relative risk of 1.12 (95% CI: 1.06, 1.19) for a 24.5 $\mu g/m^3$ increase in annual mean $PM_{2.5}$ concentrations. This corresponds to an approximate 0.5% increase in premature mortality rates per $\mu g/m^3$ increase in annual mean $PM_{2.5}$

concentrations. Alternative models based on median $PM_{2.5}$ concentrations included methods to account for spatial autocorrelation, finding a similar relative risk to the original study (1.16) but a wider confidence interval (95% CI: 0.99, 1.37). No spatial autocorrelation model was applied to mean $PM_{2.5}$ concentrations.

By applying this concentration-response function in our case study, we assume that both SO_4^{2-} and NO_3^- particles have identical toxicity as "average" fine particles within the study regions. The ACS study found slightly higher risks per unit concentration for sulfates than $PM_{2.5}$ (as did the SC cohort study[23]), with no direct information available on nitrates. A recent time-series analysis based on SC data found that fine particles from coal combustion and mobile sources were associated with premature mortality but that crustal elements were not.[28] This finding agrees with studies that have associated daily mortality and morbidity more strongly with combustion particles than with noncombustion particles.[29,30] A baseline assumption of equal toxicity appears reasonable given current information. However, this is a source of significant uncertainty, and ongoing and future research may substantially alter this assumption.

Within the HEI reanalysis,[25] the only potential confounder that demonstrated a consistent effect was SO_2 (with educational attainment acting as a strong effect modifier). The authors did not infer causality for SO_2 but, rather, stated that it could be a marker for other correlated pollutants, that the findings could be related in part to spatial patterns in air pollution, and that the bundle of $PM_{2.5}$, SO_2, and sulfates appeared to be related to premature mortality. We test concentration-response functions corresponding to all three pollutants in our sensitivity analysis.

The HEI reanalysis found no evidence of a threshold at the annual average concentrations evaluated in the study (mean $PM_{2.5}$ concentrations of 10–38 $\mu g/m^3$, mean SO_4^{2-} concentrations of 1–27 $\mu g/m^3$). By way of comparison, annual average $PM_{2.5}$ concentrations were 8–17 $\mu g/m^3$ in Massachusetts in 2000, with total particulate sulfates of 8–9 $\mu g/m^3$.[31] Thus, the concentration range in the ACS study is relevant for evaluation of health benefits in Massachusetts and nearby states, although more uncertainty exists at the lower end of the concentration ranges. Although many areas in Massachusetts are below the pending $PM_{2.5}$ annual NAAQS of 15 $\mu g/m^3$, the health literature does not provide evidence of a population threshold. The nonthreshold assumption requires additional research and is clearly a significant source of uncertainty, which we address in our sensitivity analysis.

Although compelling evidence exists for the possibility of long-term exposure effects, there are numerous uncertainties related to the interpretation of this evidence. If a cohort effect did not exist, we could alternatively consider the evidence from time-series studies, for which there are relatively more studies and fewer analytical concerns (because only other air pollutants and weather can realistically act as confounders). For this sensitivity analysis scenario, we draw our estimate from the National Morbidity, Mortality, and Air Pollution Study of the 90 largest cities in the United States,[32] which found that mortality rates increase by 0.5% for every 10 $\mu g/m^3$ increase in daily PM_{10} concentrations. This is similar to the value derived in a recent meta-analysis of the PM_{10}-mortality literature, which found a pooled value of 0.6% when controlling for the effects of correlated gaseous pollutants.[33] Because we are focusing on $PM_{2.5}$, we can convert these estimates to an incremental mortality risk of ~0.1% per $\mu g/m^3$ increase in exposure to $PM_{2.5}$, assuming a standard $PM_{2.5}/PM_{10}$ ratio of 60% and assuming that only fine particles yield health effects. This risk is similar to values reported in past $PM_{2.5}$ time-series studies.[34] It should be noted that the deaths from time-series studies are not commensurate with deaths from cohort studies, in terms of life expectancy lost and other characteristics.

At-Risk Subpopulations

For premature mortality, the determination of at-risk subpopulations has multiple components. Within our case study, we assume that the at-risk group consists of all individuals above the age of 30 (as studied in the ACS cohort), with no differential relative risk by age, gender, health status, or other demographic characteristics. We also use the U.S. average mortality rate for this age group and apply it to all census tracts in the receptor region. Clearly, a more accurate analysis would incorporate tract-specific mortality rates stratified by demographic characteristics, along with differential relative risks across demographic strata. However, there is limited evidence indicating differential relative risks for cohort mortality. As mentioned earlier, educational attainment was found to be an effect modifier of mortality in the ACS study,[25] but ecological covariates such as income, poverty, and race showed little effect. Additional evidence can be taken from recent time-series studies, which found relative risks to be relatively homogeneous across all characteristics but baseline health status.[35–37] Despite the numerous assumptions underlying our at-risk population determination, it is unclear what the magnitude or direction of any potential bias would be. The bias would be anticipated to be greater for sources in close proximity to high-risk or low-risk communities for health outcomes known to have large geographic variability (e.g., power plants near urban areas for the evaluation of asthma exacerbation or emergency room visits).

Valuation

The final component of a damage function model is generally valuation of the array of quantified impacts. For human health, this can take the form of economic valuation, medical-based metrics (such as quality-adjusted or disability-adjusted life years), or indicator metrics as used in life-cycle impact assessment. Given the substantial uncertainty both within and between valuation categories, and given that we are only quantifying one health outcome in our case study, we do not consider valuation in a substantive way in this article.

For illustrative purposes, we provide a simple benefit estimate based on the EPA value of statistical life (VSL) central estimate of $5.8 million (in 1997 dollars),[13] based on an evaluation of past wage-risk and willingness-to-pay studies. Although a detailed evaluation of the literature is beyond the scope of this article, it is worth noting that the EPA value is based on study populations (largely healthy workers) that differ somewhat from the individuals at highest risk from air pollution (likely elderly individuals with pre-existing cardiovascular or respiratory disease). For older individuals, fewer life years are at risk from current-period mortality risks, but the opportunity cost of spending on risk reduction is also lower.[38] Thus, it is not theoretically obvious what the precise consequence of this difference would be. Alternatively, valuation can be placed in life-year terms, taking advantage of the Cox proportional hazards model that implicitly provides information on the loss of life expectancy. However, the notion that all life years would be valued equally is not supported by standard economic theory.

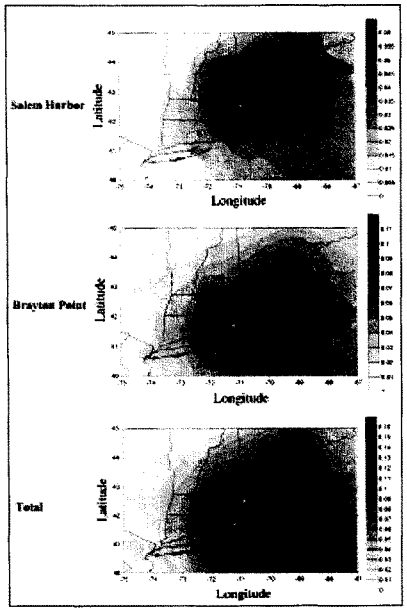

Figure 2. Geographic distribution of benefits from emission controls at the Salem Harbor and Brayton Point power plants (μg/m³ of secondary PM, annual average).

RESULTS

In this section, we provide the results of our case study analysis using our central estimates for all parameters. We evaluate the sensitivity of the estimated concentration reductions and health benefits to key assumptions in the following section. Using CALPUFF under baseline parametric assumptions, we estimate that SO_2 and NO_x emission controls at the Brayton Point and Salem Harbor power plants lead to annual average $PM_{2.5}$ reductions of 0.006–0.2 μg/m³, depending on the location in our modeling domain (see Figure 2). The benefits tend to peak in relatively close proximity to the power plants, although with some distance required for secondary particulate formation to occur. The maximum annual average benefit occurs ~40 km from the source for Salem Harbor and 25 km from the source for Brayton Point. Peaks occur at generally greater distances within seasons, but differences in wind patterns and mixing heights by season lead to annual average peaks relatively closer to the source. As would be expected, the geographic patterns of annual average concentration reductions generally follow the prevailing wind direction in New England. For both power plants, benefits 300 km toward the northeast are approximately 5–6 times greater than benefits 300 km toward the west.

Secondary sulfate particles provide a majority of benefits from both facilities. We can summarize the contribution of each pollutant by considering the population-weighted annual average concentration reduction (taking the benefit at each census tract, multiplying by the population within that tract, and dividing by the total population). Given our assumptions regarding concentration-response functions, the health benefits will be directly proportional to the population-weighted concentration reduction. Using this measure, sulfates contribute 83% of the benefits from Brayton Point and 88% of the benefits from Salem Harbor. This large SO_4^{2-} contribution is principally a function of the relative emissions of SO_2 and NO_x, as well as the fact that NH_3 preferentially reacts with SO_4^{2-} over NO_3^- and was assumed to be limited over our modeling domain. When we combine the modeled concentration reductions with our baseline $PM_{2.5}$ concentration-response function, we estimate ~70 fewer

Table 3. Implications of key assumptions on aggregate mortality risk reduction estimate for Brayton Point and Salem Harbor combined (holding other assumptions constant).

Modified Assumption	Total Mortality Risk Reduction[a]		
	Sulfates	Nitrates	Total
None (baseline)	59	11	70
Seasonally varying emissions	57	11	68
Addition of primary PM	59	11	79[b]
Dispersion modeling assumptions			
Use of RIVAD/ARM3 in CALPUFF	59	23	82
Exclusion of wet/dry deposition	83	15	97
Use of 10-ppb NH_3	59	17	76
Assumption of overestimation within 50 km, beyond 200 km of source	43	7	51
Assumption of overestimation beyond 200 km of source	48	9	58
Extension of model domain indefinitely	73	14	87
Concentration-response (C-R) assumptions			
Original ACS C-R for $PM_{2.5}$	82	15	97
HEI ACS C-R for sulfates, no NO_3^- impact	100	0	100
HEI ACS C-R for SO_2, no PM impact	–	–	290[b]
Original SC C-R for $PM_{2.5}$	160	30	190
Baseline ACS for $PM_{2.5}$, NAAQS violators only	14	2	15
Time-series $PM_{2.5}$ mortality only	14	3	16

[a]All figures are presented to two significant figures. Sums may not add due to rounding; [b]Including impacts other than sulfates and nitrates.

deaths per year (70% from Brayton Point) across a total population of 33 million and an "at-risk" population (age 30 and older) of 19 million (see Table 3). In our baseline model framework, the geographic distribution of individual health benefits is identical to the distribution of concentration reductions.

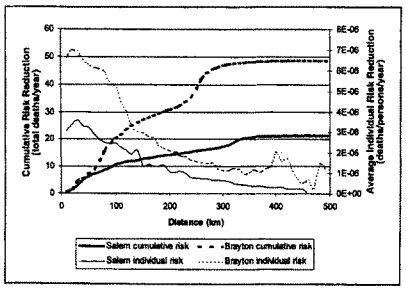

Figure 3. Distribution of individual and aggregate mortality risk reduction from emission reductions at Brayton Point and Salem Harbor power plants as a function of distance from the source.

We can plot both individual and aggregate risk reductions as a function of distance from the source to illustrate an important distributional difference (see Figure 3). In this figure, risks are averaged across all receptors in 10-km bins. Because concentration patterns and receptor locations are geographically skewed, the distribution of individual risk reductions is not always monotonic. For both power plants, individual benefits are greater closer to the facilities, given concentration patterns. However, for our modeling domain, only a small fraction of the population lives at close range (6% within 50 km of Brayton Point, 10% for Salem Harbor). Thus, a majority of the aggregate benefits accrues at long range, with more than half of the benefits found beyond 100 km of the source. This illustrates the importance of population patterns and density in determining aggregate benefits. Using the standard EPA valuation for premature mortality, the annual monetary benefits corresponding to our central health benefit estimate would be approximately $400 million. Given the substantial uncertainty in the VSL estimate, this calculation should be considered illustrative at best.

SENSITIVITY ANALYSIS

Although we presented the mortality benefits of emission controls as point estimates, as described above, the estimates are quite uncertain. While we cannot evaluate the full scope of uncertainties, we can determine the implications of a limited number of quantifiable assumptions to make a general determination of the relative magnitudes of uncertainties and the possible direction of any biases. We quantify the sensitivity of our findings to the incorporation of seasonal emissions, omission of primary PM, parametric assumptions in CALPUFF, the size of the modeling domain, and the concentration-response function for premature mortality for each of the particle constituents. We do not address monetary valuation, nor do we incorporate the assumptions listed in Table 2 that are currently unquantifiable (such as plant utilization or application of CALPUFF rather than other dispersion models). We present the implication of each assumption independently and consider discrete combinations of a limited number of assumptions. While an ideal analysis would combine estimated distributions for all parameters into a single probability density function, the accurate estimation of distributions or even probability weights to assign to different parametric assumptions is beyond the scope of our analysis.

Incorporation of Seasonal Emissions

As mentioned earlier, we modeled concentration and health benefits using both uniform and seasonally varying emissions. The findings for these analyses are essentially

identical (see Table 3). With the varying emissions model, aggregate SO_4^{2-} benefits decrease by 5% for Salem Harbor and 1% for Brayton Point, while NO_3^- benefits decrease by 3% for Salem Harbor and increase by 1% for Brayton Point. This is largely due to the similarity in emission rates and heat inputs across seasons. Our dispersion model estimated SO_4^{2-} impacts per unit emissions that were nearly an order of magnitude greater in the summer than in the winter, indicating that this factor could be important for sources with significant seasonality in emissions.

Omission of Primary Particulate Matter

Although we have omitted filterable and condensable PM from our baseline analysis due to the structure of proposed regulations in Massachusetts, both pollutants could be significant contributors to health benefits and would be affected by on-site control measures. To determine baseline filterable $PM_{2.5}$, we gathered data on 1996–1998 average PM_{10} emission rates and used EPA's Particle Calculator Version 2.0.2[39] to estimate the $PM_{2.5}/PM_{10}$ ratio. Lower target PM_{10} under BACT was determined to be 0.01 lb/MMBTU. For simplicity, we assumed that the $PM_{2.5}/PM_{10}$ ratio would be unchanged from current levels (which likely provides an upper bound on omitted benefits, because many control measures disproportionately decrease larger particles). For current condensable PM, we used AP-42 emission factors given coal sulfur content. Lower target levels were taken from AP-42 estimates for facilities with control technology in place, and all condensable PM was assumed to be in the fine fraction.

Under these assumptions, the mortality benefits associated with primary $PM_{2.5}$ emission reductions would be on the order of 10 fewer deaths per year, increasing our baseline estimate by 13% (see Table 3). As anticipated, the near-source contribution is more substantial than for secondary PM, with 32% of primary $PM_{2.5}$ benefits for Brayton Point and 60% for Salem Harbor occurring within 50 km of the source (versus 15 and 29% for secondary PM, respectively). Thus, our omission of primary $PM_{2.5}$ slightly underestimates the benefits of control and the proportion of near-source benefits.

Parametric Uncertainty in CALPUFF

In our sensitivity analysis, we test three major parametric assumptions in CALPUFF—the chemical conversion mechanism, the incorporation of wet/dry deposition, and the background NH_3 concentration. When we use the RIVAD/ARM3 chemical mechanism rather than MESOPUFF II (holding all else constant), SO_4^{2-} benefits are essentially unchanged (5% decrease at Brayton Point, 12% increase at Salem Harbor), but NO_3^- benefits are increased substantially (doubled at both power plants). The MESOPUFF II chemical mechanism is generally preferred (particularly in urban settings), but this demonstrates the model sensitivity of NO_3^- benefit estimates relative to SO_4^{2-} benefit estimates. Because a majority of total benefits are related to sulfates, the choice of chemical mechanism has a relatively small influence on total benefits (see Table 3).

We did not evaluate whether our incorporation of deposition might have overestimated benefits (insufficient deposition), but we can place a bound on any potential underestimation by removing all deposition terms from the model. This would clearly overestimate benefits, but given the numerous uncertainties associated with deposition rates and scavenging coefficients[15] and the omission of risks associated with the deposited materials, this may not be an inappropriate conservative estimate. Omitting deposition increases benefits for both sulfates (40% increase at Brayton Point and 39% increase at Salem Harbor) and nitrates (34% increase at Brayton Point and 29% increase at Salem Harbor). Finally, increasing the background NH_3 concentration to the CALPUFF default of 10 ppb has no effect on sulfates but increases NO_3^- benefits by 54% at Brayton Point and 45% at Salem Harbor. Thus, perturbing these three parametric assumptions tends to increase benefits, with atmospheric chemistry assumptions influencing nitrates more than sulfates (see Table 3).

Size of the Modeling Domain

Any uncertainty associated with our modeling domain is difficult to quantify, because we do not have the data to directly quantify long-range concentrations. Our modeling domain could overestimate benefits if CALPUFF were upwardly biased at longer range, or it could yield an underestimate if a significant fraction of exposure occurred beyond the boundaries of our receptor region. Tracer dispersion experiments found that CALPUFF was unbiased between 50 and 200 km, but EPA found that CALPUFF might overestimate at long range by as much as a factor of 2.[17] A reasonable lower bound would assume that all concentrations beyond 200 km were overestimated by a factor of 2 and, because near-source (<50 km) performance was not evaluated, that a similar bias exists there. This would reduce benefits by 26% for Brayton Point and 31% for Salem Harbor. If we only consider the possibility of overestimation beyond 200 km, these figures are reduced to 18 and 16% (see Table 3).

To place a bound on any potential underestimation due to our limited receptor region, we can fit a simple regression between concentration reductions and distance from the source. Beyond 50 km, total secondary PM concentration reductions can be well predicted as an exponential function of distance ($R^2 = 0.81$ for Brayton Point and 0.87 for Salem Harbor). If we assume for simplicity that population density is uniform at long range, we can

determine the degree of underestimation due to our abbreviated model domain. In total, we estimate that increasing our modeling bounds indefinitely would increase benefits by ~19% for Salem Harbor and 26% for Brayton Point (Table 3). Thus, it does not appear that we have significantly underestimated benefits, potentially related in part to the preponderance of long-range receptors at largely upwind locations.

Concentration-Response Function

There are clearly numerous plausible estimates of mortality concentration-response functions, based on reported confidence intervals, alternative statistical models within studies, use of different studies, and alternative assumptions about particle constituent toxicity. Although evaluating the complete range of uncertainties is beyond the scope of this paper, we can consider a set of discrete scenarios as alternatives to our baseline concentration-response function. These scenarios are listed next, with the figure in parentheses indicating the central estimate of the mortality increase per $\mu g/m^3$ of concentration increase.

- the original reported ACS concentration-response function for $PM_{2.5}$ (0.6%);[22]
- ACS concentration-response function for sulfates (0.8%, Table 31 of HEI reanalysis[25]), with no health impacts from nitrates;
- ACS concentration-response function for SO_2 in multivariate models (0.5%, Table 50 of HEI reanalysis[25]), assuming that SO_2 is a more appropriate marker than $PM_{2.5}$;
- our baseline ACS concentration-response function for $PM_{2.5}$ (0.5%), applied only to receptors in counties with any monitors exceeding the pending annual NAAQS (15 $\mu g/m^3$, 1999 data);
- the original reported SC concentration-response function for $PM_{2.5}$ (1.2%);[23] and
- zero $PM_{2.5}$ cohort mortality effect (as implied by VA[26] or selected models from other studies), with a time-series effect of 0.1%.

Depending on the concentration-response assumption, our total benefits can vary significantly from our baseline estimate. If we assume either that cohort mortality effects are not present or that they are only applicable for counties exceeding the pending annual $PM_{2.5}$ NAAQS, our benefit estimates are decreased substantially. On the other hand, using the SC concentration-response function or assuming that the SO_2 concentration-response function from the ACS reanalysis is appropriate increases our benefits significantly (see Table 3). More moderate differences are associated with using the original ACS concentration-response function or considering impacts to only be associated with SO_4^{2-} particles (39 and 44% increases in benefits, respectively).

Sensitivity Propagation

The above calculations illustrate that our total benefit estimates are sensitive to key parametric assumptions, with the largest quantifiable influence associated with the assumed concentration-response function. To provide a sense of the range of benefit estimates implied by quantifiable uncertainties, we consider discrete combinations of a subset of factors. While this should not be construed as a formal uncertainty analysis, it can help place some of the parametric uncertainties in context. If we combine the lower bound concentration-response functions with an assumption of dispersion modeling overestimation outside the 50–200-km range, our benefit estimate would be on the order of 10 deaths per year. An extreme upper bound would use the SO_2 concentration-response function with no deposition and an indefinite model domain, yielding a benefit of ~400 deaths per year. We consider both of these estimates to be unlikely and not representative of best modeling practice or literature interpretation. Excluding the SO_2 mortality scenario as well as the scenarios where no cohort effect exists or it only exists at more than 15 $\mu g/m^3$, we find benefit estimates that range between ~50 and 200 premature deaths per year.

As a comparison point, we can also calculate uncertainty bounds by conventional propagation of uncertainties as determined by 95% confidence intervals surrounding our baseline model estimate. A typical assumption would be that the population-weighted annual average concentrations have a 95% confidence interval between 50 and 150% of the central estimate (an interval generally applied to the Industrial Source Complex model[40] for shorter-term measurements in single locations). We combine this with the 95% confidence interval for our baseline premature mortality estimate, as reported in the HEI reanalysis (95% CI: 1.06, 1.19).[21] When we combine these two confidence intervals using Monte Carlo analysis, we find a mean mortality risk reduction of 70 deaths per year (95% CI: 30, 120). This clearly does not incorporate the range of issues within our sensitivity analysis but does encompass a number of the simple sensitivity estimates in Table 3.

DISCUSSION

Our case study has demonstrated that the magnitude and distribution of health benefits of power plant emission controls can be estimated reasonably. Through the application of an atmospheric dispersion model coupled with epidemiologic evidence regarding the health benefits of incremental concentration reductions, our central estimate is that requiring two Massachusetts power plants to apply BACT would lead to ~70 fewer premature deaths per year over a broad region. There are numerous obstacles in the interpretation of these findings. Although we likely

captured a significant contributor to benefits, without quantification of control costs or other benefits, it is difficult to interpret the importance of the findings. In addition, the individual risk reduction is relatively small, and the $PM_{2.5}$ concentration reductions are on the order of 2% of ambient concentrations. However, it is important to recognize that we have modeled only secondary PM from a subset of sources from one sector within one state. Any such analysis would find a relatively small contribution to ambient concentrations, but this does not imply that regulatory action would not be justified. This determination requires a comparison between the full array of benefits and the full array of control costs. The more difficult related issue is the potential increase in atmospheric modeling uncertainty for small concentration increments.

We have also only quantified a limited degree of uncertainty, and additional elements in the atmospheric modeling and health evidence (as well as plant utilization and other dimensions not addressed in our case study) could significantly affect our benefit estimates. For the dispersion modeling, our parametric sensitivity analysis lends support to the relative robustness of our estimate with respect to changes internal to CALPUFF. However, CALPUFF may be biased when compared with other models. For example, issues related to CALPUFF's aqueous-phase chemistry for SO_4^{2-} formation have been raised, which may imply significant underestimation of SO_4^{2-} formation and impacts.[17] We can validate our findings to a limited degree by comparing our analysis with other studies that used alternative dispersion models but made similar assumptions elsewhere.

For example, a recent study by Abt Associates[43] used REMSAD and a source-receptor (S-R) matrix to determine the benefits of a 75% emission reduction of SO_2 and NO_x from all power plants in the United States. Using the identical concentration-response function as in our baseline model, they determined annual benefits of ~19,000 fewer deaths per year using REMSAD and 12,000 fewer deaths per year using S-R from annual emission reductions of 7 million tons of SO_2 and 2 million tons of NO_x. The emission reductions in our study are ~0.8% of the national SO_2 reduction and 0.6% of the NO_x reduction. Applying these ratios to the above mortality benefits yields numbers on an order of magnitude of 100 fewer deaths per year, similar to our finding. While this is far from direct validation of the CALPUFF model and its application, the similarity of our results to those using other models lends plausibility to the order of magnitude of our findings.

Considering the health literature, our extreme assumptions demonstrated that there are some substantial uncertainties. For one, we have assumed that the cohort mortality evidence reflects a causal relationship for $PM_{2.5}$ that can be attributed solely to $PM_{2.5}$. We have also assumed that the slope of the concentration-response curve at current ambient levels is similar to the slope derived in the ACS cohort study. Both assumptions reflect scientific questions for which uncertainty may be reduced by future research. However, one of the advantages of the damage function framework is that it can transparently provide benefit estimates under a number of scenarios. Thus, decision-makers can take their beliefs for key parameters (e.g., the existence of health benefits below the NAAQS for $PM_{2.5}$) and determine the corresponding benefits, or they can use the range of values to determine if their policy decisions are influenced by selected assumptions.

Aside from the specific evidence we used, two broad critiques can be raised about the damage function/environmental externality approach. Related to some of the uncertainties listed previously, there is the argument that current knowledge about important model components (i.e., atmospheric chemistry, relative toxicity of particulate constituents, biological mechanisms supporting causality) is insufficient to construct damage function models. In other words, even if we could construct more detailed uncertainty analyses, we simply do not know enough about the behavior of air pollutants to begin to quantify the health benefits of air pollution control. While it is true that significant scientific uncertainties exist and will continue to exist for the foreseeable future, this should not act as a barrier to action or analysis. Rather, this implies that researchers should carefully analyze the range of uncertainties and determine whether the uncertainties might materially affect policy choices. In addition, the scientific uncertainties must be placed in context. For example, a dispersion model that does a poor job estimating the precise location of concentration peaks but accurately estimates population-weighted annual average concentrations is quite useful for our application.

A second concern is that the damage function approach (and the corresponding benefit-cost analyses that could be conducted) is contrary to the current regulatory structure for criteria pollutants in the United States, which focuses largely on the establishment of NAAQS and the development of plans to avoid violations. Thus, damage function modeling may be reasonably accurate, but it does not provide information relevant to policy-makers. While this is correct on its face, we would assert that the damage function approach has a number of applications within the existing regulatory framework. For example, as emission control plans are developed, damage function modeling can determine the magnitude and distribution of health benefits from an array of policies that might all achieve NAAQS compliance. Policy-makers could then select a portfolio of options that achieves the identical regulatory purpose at minimum cost with maximum aggregate benefits and reduced environmental inequities.

In addition, when multiple source categories and pollutants are modeled in a similar framework, damage function modeling can be used to help in overall prioritization of future research and regulatory agendas.

Future analyses should focus on incorporating the remaining elements necessary for an adequate benefit-cost analysis and on generalizing our findings to other settings. To have relevance for pending policy decisions, our damage function model should be applied to evaluate the benefits of specific proposed regulations (e.g., on-site emission reduction to BACT levels, unrestricted emission trading, mandatory partial on-site reductions coupled with regional emission trading). Ozone, Hg, CO_2, and any other pollutants associated with the control measures should be included if shown to be important, and the economic implications of the regulations should be ascertained. Regarding generalizability, while some meteorological and topographic characteristics are unique to Massachusetts, it is clear that our findings can be extrapolated to a limited extent to other settings (particularly those in close proximity to the modeled facilities). Furthermore, recent work has demonstrated that the exposure per unit emissions from a power plant is reasonably invariant across plants for secondary particles[42] and can be predicted well by a limited number of parameters (such as population density and climate).[43] Additional studies should confirm these relationships for different source types and settings.

CONCLUSIONS

We have constructed a model to quantify the concentration and health benefits associated with NO_x and SO_2 emission reductions from power plants. Application of our model to two power plants in Massachusetts finds a reduction of ~70 premature deaths per year associated with decreases in secondary PM concentrations. Although further research would be needed to incorporate additional elements in our model (including future plant utilization and life-cycle emissions) and to more comprehensively characterize uncertainties, our findings are relatively robust with respect to parametric changes in the dispersion model or moderate changes in assumed concentration-response functions. Given the potential magnitude of health benefits from large-scale regulation of power plants or other significant emission sources, the damage function modeling approach should be used in conjunction with cost information to inform future control strategies.

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ORIGINAL CONTRIBUTION

Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution

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Context Associations have been found between day-to-day particulate air pollution and increased risk of various adverse health outcomes, including cardiopulmonary mortality. However, studies of health effects of long-term particulate air pollution have been less conclusive.

Objective To assess the relationship between long-term exposure to fine particulate air pollution and all-cause, lung cancer, and cardiopulmonary mortality.

Design, Setting, and Participants Vital status and cause of death data were collected by the American Cancer Society as part of the Cancer Prevention II study, an ongoing prospective mortality study, which enrolled approximately 1.2 million adults in 1982. Participants completed a questionnaire detailing individual risk factor data (age, sex, race, weight, height, smoking history, education, marital status, diet, alcohol consumption, and occupational exposures). The risk factor data for approximately 500 000 adults were linked with air pollution data for metropolitan areas throughout the United States and combined with vital status and cause of death data through December 31, 1998.

Main Outcome Measure All-cause, lung cancer, and cardiopulmonary mortality.

Results Fine particulate and sulfur oxide–related pollution were associated with all-cause, lung cancer, and cardiopulmonary mortality. Each 10-μg/m³ elevation in fine particulate air pollution was associated with approximately a 4%, 6%, and 8% increased risk of all-cause, cardiopulmonary, and lung cancer mortality, respectively. Measures of coarse particle fraction and total suspended particles were not consistently associated with mortality.

Conclusion Long-term exposure to combustion-related fine particulate air pollution is an important environmental risk factor for cardiopulmonary and lung cancer mortality.

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Based on several severe air pollution events,[1-3] a temporal correlation between extremely high concentrations of particulate and sulfur oxide air pollution and acute increases in mortality was well established by the 1970s. Subsequently, epidemiological studies published between 1989 and 1996 reported health effects at unexpectedly low concentrations of particulate air pollution.[4] The convergence of data from these studies, while controversial,[5] prompted serious reconsideration of standards and health guidelines[6-10] and led to a long-term research program designed to analyze health-related effects due to particulate pollution.[11-13] In 1997, the Environmental Protection Agency adopted new ambient air quality standards that would impose regulatory limits on fine particles measuring less than 2.5 μm in diameter ($PM_{2.5}$). These new standards were challenged by industry groups, blocked by a federal appeals court, but ultimately upheld by the US Supreme Court.[14]

Although most of the recent epidemiological research has focused on effects of short-term exposures, several studies suggest that long-term exposure may be more important in terms of overall public health.[4] The new standards for long-term exposure to $PM_{2.5}$ were originally based primarily on 2 prospective cohort studies,[15,16] which evaluated the effects of long-term pollution exposure on mortality. Both of these studies have been subjected to much scrutiny,[5] including an extensive independent audit and reanalysis of the original data.[17] The larger of these 2 studies linked individual risk factor and vital status data with national ambient air pollution data.[16] Our analysis uses data from the larger study and

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(1) doubles the follow-up time to more than 16 years and triples the number of deaths; (2) substantially expands exposure data, including gaseous copollutant data and new $PM_{2.5}$ data, which have been collected since the promulgation of the new air quality standards; (3) improves control of occupational exposures; (4) incorporates dietary variables that account for total fat consumption, and consumption of vegetables, citrus, and high-fiber grains; and (5) uses recent advances in statistical modeling, including the incorporation of random effects and nonparametric spatial smoothing components in the Cox proportional hazards model.

METHODS

Study Population

The analysis is based on data collected by the American Cancer Society (ACS) as part of the Cancer Prevention Study II (CPS-II), an ongoing prospective mortality study of approximately 1.2 million adults.[18,19] Individual participants were enrolled by ACS volunteers in the fall of 1982. Participants resided in all 50 states, the District of Columbia, and Puerto Rico, and were generally friends, neighbors, or acquaintances of ACS volunteers. Enrollment was restricted to persons who were aged 30 years or older and who were members of households with at least 1 individual aged 45 years or older. Participants completed a confidential questionnaire, which included questions about age, sex, weight, height, smoking history, alcohol use, occupational exposures, diet, education, marital status, and other characteristics.

Vital status of study participants was ascertained by ACS volunteers in September of the following years: 1984, 1986, and 1988. Reported deaths were verified with death certificates. Subsequently, through December 31, 1998, vital status was ascertained through automated linkage of the CPS-II study population with the National Death Index.[19] Ascertainment of deaths was more than 98% complete for the period of 1982-1988 and 93% complete after 1988.[19] Death certificates or codes for cause of death were obtained for more than 98% of all known deaths. Cause of death was coded according to the *International Classification of Diseases, Ninth Revision (ICD-9)*. Although the CPS-II cohort included approximately 1.2 million participants with adequate questionnaire and cause-of-death data, our analysis was restricted to those participants who resided in US metropolitan areas with available pollution data. The actual size of the analytic cohort varied depending on the number of metropolitan areas for which pollution data were available. TABLE 1 provides the number of metropolitan areas and participants available for each source of pollution data.

Air Pollution Exposure Estimates

Each participant was assigned a metropolitan area of residence based on address at time of enrollment and 3-digit ZIP code area.[20] Mean (SD) concentrations of air pollution for the metropolitan areas were compiled from various primary data sources (Table 1). Many of the particulate pollution indices, including $PM_{2.5}$, were available from data from the Inhalable Particle Monitoring Network for 1979-1983 and data from the National Aerometric Database for 1980-1981, periods just prior to or at the beginning of the follow-up period. An additional data source was the Environmental Protection Agency Aerometric Information Retrieval System (AIRS). The mean concentration of each pollutant from all available monitoring sites was calculated for each metropolitan area during the 1 to 2 years prior to enrollment.[17]

Additional information on ambient pollution during the follow-up period was extracted from the AIRS database as quarterly mean values for each routinely monitored pollutant for 1982 through 1998. All quarterly averages met summary criteria imposed by the Environmental Protection Agency and were based on observations made on at least 50% of the scheduled sampling days at each site. The quarterly mean values for all stations in each metropolitan area were calculated across the study years using daily average values for each pollutant except ozone. For ozone, daily 1-hour maximums were used and were calculated for the full year and for the third quarter only (ie, July, August, September). While gaseous pollutants generally had recorded data throughout the entire follow-up period of interest, the particulate matter monitoring protocol changed in the late 1980s from total suspended particles to particles measuring less than 10 μm in diameter (PM_{10}), resulting in the majority of total suspended particle data being available in the early to mid-1980s and PM_{10} data being mostly available in the early to mid-1990s.

As a consequence of the new $PM_{2.5}$ standard, a large number of sites began collecting $PM_{2.5}$ data in 1999. Daily $PM_{2.5}$ data were extracted from the AIRS database for 1999 and the first 3 quarters of 2000. For each site, quarterly averages for each of the 2 years were computed. The 4 quarters were averaged when at least 1 of the 2 corresponding quarters for each year had at least 50% of the sixth-day samples and at least 45 total sampling days available. Measurements were averaged first by site and then by metropolitan area. Although no network of $PM_{2.5}$ monitoring existed in the United States between the early 1980s and the late 1990s, the integrated average of $PM_{2.5}$ concentrations during the period was estimated by averaging the $PM_{2.5}$ concentration for early and later periods.

Mean sulfate concentrations for 1980-1981 were available for many cities based on data from the Inhalable Particle Monitoring Network and the National Aerometric Database. Recognizing that sulfate was artifactually overestimated due to glass fiber filters used at that time, season and region-specific adjustments were made.[17] Since few states analyzed particulate samples for sulfates after the early 1980s, individual states were directly contacted for data regarding filter use. Ion chromatography was used to analyze PM_{10} filters and this data could be obtained from metropolitan areas across the

United States. Filters were collected for a single reference year (1990) in the middle of the 1982-1998 study period. The use of quartz filters virtually eliminated the historical overestimation of sulfate. Mean sulfate concentrations for 1990 were estimated using sulfate from AIRS, data reported directly from individual states, and analysis of archived filters.

Statistical Analysis

The basic statistical approach used in this analysis is an extension of the standard Cox proportional hazards survival model,[21] which has been used for risk estimates of pollution-related mortality in previous longitudinal cohort studies.[15,16] The standard Cox model implicitly assumes that observations are statistically independent after controlling for available risk factors, resulting in 2 concerns with regard to risk estimates of pollution-related mortality.[22] First, if the assumption of statistical independence is not valid, the uncertainty in the risk estimates of pollution-related mortality may be misstated. Second, even after controlling for available risk factors, survival times of participants living in communities closer together may be more similar than participants living in communities farther apart, which results in spatial autocorrelation. If this spatial autocorrelation is due to missing or systematically mismeasured risk factors that are spatially correlated with air pollution, then the risk estimates of pollution-related mortality may be biased due to inadequate control of these factors. Therefore, in this analysis, the Cox proportional hazards model was extended by incorporating a spatial random-effects component, which provided accurate es-

Table 1. Summary of Alternative Pollution Indices*

Pollutant (Years of Data Collection)	Units	Source of Data	Data Compilation Team†	No. of Metropolitan Areas	No. of Participants, in Thousands	Mean (SD)
$PM_{2.5}$	$\mu g/m^3$					
1979-1983		IPMN	HEI	61	359	21.1 (4.6)
1999-2000		AIRS	NYU	116	500	14.0 (3.0)
Average				51	319	17.7 (3.7)
PM_{10}	$\mu g/m^3$					
1982-1998		AIRS	NYU	102	415	28.8 (5.9)
PM_{15}	$\mu g/m^3$					
1979-1983		IPMN	HEI	63	359	40.3 (7.7)
$PM_{15-2.5}$	$\mu g/m^3$					
1979-1983		IPMN	HEI	63	359	19.2 (6.1)
Total suspended particles	$\mu g/m^3$					
1980-1981		NAD	HEI	156	590	68.0 (16.7)
1979-1983		IPMN	HEI	58	351	73.7 (14.3)
1982-1998		AIRS	NYU	150	573	56.7 (13.1)
Sulfate	$\mu g/m^3$					
1980-1981		IPMN and NAD, artifact adjusted	HEI	149	572	6.5 (2.8)
1990		Compilation and analysis of PM_{10} filters	NYU	53	269	6.2 (2.0)
Sulfur dioxide	ppb	AIRS				
1980			HEI	118	520	9.7 (4.9)
1982-1998			NYU	126	539	6.7 (3.0)
Nitrogen dioxide	ppb	AIRS				
1980			HEI	78	409	27.9 (9.2)
1982-1998			NYU	101	493	21.4 (7.1)
Carbon monoxide	ppm	AIRS				
1980			HEI	113	519	1.7 (0.7)
1982-1998			NYU	122	536	1.1 (0.4)
Ozone	ppb	AIRS				
1980			HEI	134	569	47.9 (11.0)
1982-1998			NYU	119	525	45.5 (7.3)
1982-1998‡			NYU	134	557	59.7 (12.8)

*$PM_{2.5}$ indicates particles measuring less than 2.5 μm in diameter; PM_{10}, particles measuring less than 10 μm in diameter; PM_{15}, particles measuring less than 15 μm in diameter; $PM_{15-2.5}$, particles measuring between 2.5 and 15 μm in diameter; $\mu g/m^3$, micrograms per cubic meter; ppb, parts per billion; ppm, parts per million; IPMN, Inhalable Particle Monitoring Network; AIRS, Aerometric Information Retrieval System [Environmental Protection Agency]; and NAD, National Aerometric Database.
†HEI indicates data were compiled by the Health Effects Institute reanalysis team, which was previously published.[17] NYU indicates data were compiled at the New York University School of Medicine, Nelson Institute of Environmental Medicine (K.I. and G.D.T.).
‡Daily 1-hour maximums were used. Values were calculated only for the third quarter (ie, July, August, September).

timates of the uncertainty of effect estimates. The model also evaluated spatial autocorrelation and incorporated a nonparametric spatial smooth component (to account for unexplained spatial structure). A more detailed description of this modeling approach is provided elsewhere.[22]

The baseline analysis in this study estimated adjusted relative risk (RR) ratios for mortality by using a Cox proportional hazards model with inclusion of a metropolitan-based random-effects component. Model fitting involved a 2-stage process. In the first stage, survival data were modeled using the standard Cox proportional hazards model, including individual level covariates and indicator variables for each metropolitan area (without pollution variables). Output from stage 1 provided estimates of the metropolitan-specific logarithm of the RRs of mortality (relative to an arbitrary reference community), which were adjusted for individual risk factors. The correlation between these values, which was induced by using the same reference community, was then removed.[23] In the second stage, the estimates of adjusted metropolitan-specific health responses were related to fine particulate air pollution using a linear random-effects regression model.[24] The time variable used in the models was survival time from the date of enrollment. Survival times of participants who did not die were censored at the end of the study period. To control for age, sex, and race, all of the models were stratified by 1-year age categories, sex, and race (white vs other), which allowed each category to have its own baseline hazard. Models were estimated for all-cause mortality and for 3 separate mortality categories: cardiopulmonary (*ICD-9* 401-440 and 460-519), lung cancer (*ICD-9* 162), and all others.

Models were estimated separately for each of the 3 fine particle variables, $PM_{2.5}$ (1979-1983), $PM_{2.5}$ (1999-2000), and $PM_{2.5}$ (average). Individual level covariates were included in the models to adjust for various important individual risk factors. All of these variables were classified as either indicator (ie, yes/no, binary, dummy) variables or continuous variables. Variables used to control for tobacco smoke, for example, included both indicator and continuous variables. The smoking indicator variables included: current cigarette smoker, former cigarette smoker, and a pipe or cigar smoker only (all vs never smoking) along with indicator variables for starting smoking before or after age 18 years. The continuous smoking variables included: current smoker's years of smoking, current smoker's years of smoking squared, current smoker's cigarettes per day, current smoker's cigarettes per day squared, former smoker's years of smoking, former smoker's years of smoking squared, former smoker's cigarettes per day, former smoker's cigarettes per day squared, and the number of hours per day exposed to passive cigarette smoke.

To control for education, 2 indicator variables, which indicated completion of high school or education beyond high school, were included. Marital status variables included indicator variables for single and other vs married. Both body mass index (BMI) values and BMI values squared were included as continuous variables. Indicator variables for beer, liquor, and wine drinkers and nonresponders vs nondrinkers were included to adjust for alcohol consumption. Occupational exposure was controlled for using various indicator variables: regular occupational exposure to asbestos, chemicals/acids/solvents, coal or stone dusts, coal tar/pitch/asphalt, diesel engine exhaust, or formaldehyde, and additional indicator variables that indicated 9 different rankings of an occupational dirtiness index that has been developed and described elsewhere.[17,25] Two diet indices that accounted for fat consumption and consumption of vegetables, citrus, and high-fiber grains were derived based on information given in the enrollment questionnaire.[18] Quintile indicator variables for each of these diet indices were also included in the models.[18]

In addition to the baseline analysis, several additional sets of analysis were conducted. First, to more fully evaluate the shape of the concentration-response function, a robust locally weighted regression smoother[26] (within the generalized additive model framework[27]) was used to estimate the relationship between particulate air pollution and mortality in the second stage of model fitting. Second, the sensitivity of the fine particle mortality risk estimates compared with alternative modeling approaches and assumptions was evaluated. Standard Cox proportional hazards models were fit to the data including particulate air pollution as a predictor of mortality and sequentially adding (in a controlled forward stepwise process) groups of variables to control for smoking, education, marital status, BMI, alcohol consumption, occupational exposures, and diet.

In addition, to evaluate the sensitivity of the estimated pollution effect while more aggressively controlling for spatial differences in mortality, a 2-dimensional term to account for spatial trends was added to the models and was estimated using a locally weighted regression smoother. The "span" parameter, which controls the complexity of the surface smooth, was set at 3 different settings to allow for increasingly aggressive fitting of the spatial structure. These included a default span of 50%, the span that resulted in the lowest unexplained variance in mortality rate between metropolitan areas, and the span that resulted in the strongest evidence (highest P value) to suggest no residual spatial structure. The risk estimates and SEs (and thus the confidence intervals) were estimated using generalized additive modeling[27] with S-Plus statistical software,[28] which provides unbiased effect estimates, but may underestimate SEs if there is significant spatial autocorrelation and significant correlations between air pollution and the smoothed surface of mortality. Therefore, evidence of spatial autocorrelation was carefully evaluated and tested using the Bartlett test.[29] The correlations of residual mortality

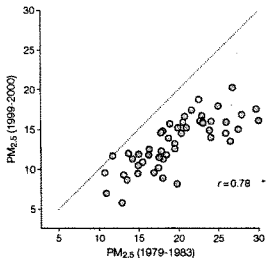
with distance between metropolitan areas were graphically examined.

Analyses were also conducted of effect modification by age, sex, smoking status, occupational exposure, and education. Finally, models were fit using a variety of alternative pollution indices, including gaseous pollutants. Specifically, models were estimated separately for each of the pollution variables listed in Table 1, while also including all of the other risk factor variables.

RESULTS

Fine particulate air pollution generally declined in the United States during the follow-up period of this study. FIGURE 1 plots mean $PM_{2.5}$ concentrations for 1999-2000 over mean $PM_{2.5}$ concentrations for 1979-1983 for the 51 cities in which paired data were available. The concentrations of $PM_{2.5}$ were lower in 1999-2000 than in 1979-1983 for most cities, with the largest reduction observed in the cities with the highest concentrations of pollution during 1979-1983. Mean $PM_{2.5}$ levels in the 2 periods were highly correlated (r=0.78). The rank ordering of cities by relative pollution levels remained nearly the same. Therefore, the relative levels of fine particle concentrations were similar whether based on measurements at the beginning of the study period, shortly following the study period, or an average of the 2.

Figure 1. Mean Fine Particles Measuring Less Than 2.5 μm in Diameter ($PM_{2.5}$)



Mean $PM_{2.5}$ concentrations in micrograms per meters cubed for 1999-2000 are plotted along with concentrations for 1979-1983 for the 51 metropolitan areas with paired pollution data. The dotted line is a reference 45°-equality line.

As reported in TABLE 2, all 3 indices of fine particulate air pollution were associated with all-cause, cardiopulmonary, and lung cancer mortality, but not mortality from all other causes combined. FIGURE 2 presents the nonparametric smoothed exposure response relationships between cause-specific mortality and $PM_{2.5}$ (average). The log RRs for all-cause, cardiopulmonary, and lung cancer mortality increased across the gradient of fine particulate matter. Goodness-of-fit tests indicated that the associations were not significantly different from linear associations (P>.20).

The fine particle mortality RR ratios from various alternative modeling approaches and assumptions are presented in FIGURE 3. After controlling for smoking, education, and marital status, the controlled forward stepwise inclusion of additional covariates had little influence on the estimated associations with fine particulate air pollution on cardiopulmonary and lung cancer mortality. As expected, cigarette smoking was highly significantly associated with elevated risk of all-cause, cardiopulmonary, and lung cancer mortality (P<.001). Estimated RRs for an average current smoker (men and women combined, 22 cigarettes/day for 33.5 years, with initiation before age 18 years) were equal to 2.58, 2.89, and 14.80 for all-cause, cardiopulmonary, and lung cancer mortality, respectively. Statistically significant, but substantially smaller and less robust associations, were also observed for education, marital status, BMI, alcohol consumption, occupational exposure, and diet variables. Although many of these covariates were also statistically associated with mortality, the risk estimates of pollution-related mortality were not highly sensitive to the inclusion of these additional covariates.

Figure 3 also demonstrates that the introduction of the random-effects component to the model resulted in larger SEs of the estimates and, therefore, somewhat wider 95% confidence intervals. There was no evidence of statistically significant spatial autocorrelation in the survival data based on the Bartlett test (P>.20) after controlling for fine particulate air pollution and the various individual risk factors. Furthermore, graphical examination of the correlations of the residual mortality with distance between metropolitan areas did not reveal significant spatial autocorrelation (results not shown). Nevertheless, the incorporation of spatial smoothing was included to further investigate the robustness of the estimated particulate pollution effect. Effect estimates were not highly sensitive to the incorporation of spatial smoothing to account for regional clustering or other spatial patterns in the data.

FIGURE 4 presents fine particle air pollution–related mortality RR ratios after stratifying by age, sex, education, and smoking status, and adjusting for all other risk factors. The differences across age and sex strata were not generally consistent or statistically significant. However, a consistent pattern emerged from this stratified analysis: the association with particulate pollution was stronger for both cardiopulmo-

Table 2. Adjusted Mortality Relative Risk (RR) Associated With a 10-μg/m³ Change in Fine Particles Measuring Less Than 2.5 μm in Diameter

	Adjusted RR (95% CI)*		
Cause of Mortality	1979-1983	1999-2000	Average
All-cause	1.04 (1.01-1.08)	1.06 (1.02-1.10)	1.06 (1.02-1.11)
Cardiopulmonary	1.06 (1.02-1.10)	1.08 (1.02-1.14)	1.09 (1.03-1.16)
Lung cancer	1.08 (1.01-1.16)	1.13 (1.04-1.22)	1.14 (1.04-1.23)
All other cause	1.01 (0.97-1.05)	1.01 (0.97-1.06)	1.01 (0.95-1.06)

*Estimated and adjusted based on the baseline random-effects Cox proportional hazards model, controlling for age, sex, race, smoking, education, marital status, body mass, alcohol consumption, occupational exposure, and diet. CI indicates confidence interval.

nary and lung cancer mortality for participants with less education. Also, for both cardiopulmonary and lung cancer mortality, the RR estimates were higher for nonsmokers.

FIGURE 5 summarizes the associations between mortality risk and air pollutant concentrations listed in Table 1. Statistically significant and relatively consistent mortality associations existed for all measures of fine particulate exposure, including $PM_{2.5}$ and sulfate particles. Weaker less consistent mortality associations were observed with PM_{10} and PM_{15}. Measures of the coarse particle fraction ($PM_{15\text{-}2.5}$) and total suspended particles were not consistently associated with mortality. Of the gaseous pollutants, only sulfur dioxide was associated with elevated mortality risk. Interestingly, measures of $PM_{2.5}$ were associated with all-cause cardiopulmonary, and lung cancer mortality, but not with all other mortality. However, sulfur oxide pollution (as measured by sulfate particles and/or sulfur dioxide) was significantly associated with mortality from all other causes in addition to all-cause, cardiopulmonary, and lung cancer mortality.

COMMENT

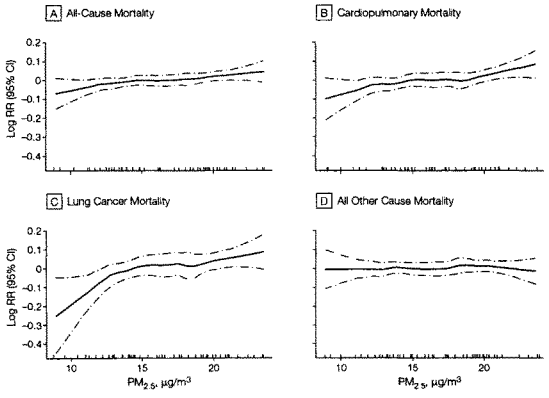
This study demonstrated associations between ambient fine particulate air pollution and elevated risks of both cardiopulmonary and lung cancer mortality. Each 10-$\mu g/m^3$ elevation in long-term average $PM_{2.5}$ ambient concentrations was associated with approximately a 4%, 6%, and 8% increased risk of all-cause, cardiopulmonary, and lung cancer mortality, respectively, although the magnitude of the effect somewhat depended on the time frame of pollution monitoring. In addition, this analysis addresses many of the important questions concerning the earlier, more limited analysis of the large CPS-II cohort, including the following issues.

First, does the apparent association between pollution and mortality persist with longer follow-up and as the cohort ages and dies? The present analysis more than doubled the follow-up time to more than 16 years, resulting in approximately triple the number of deaths, yet the associations between pollution and mortality persisted.

Second, can the association between fine particulate air pollution and increased cardiopulmonary and lung cancer mortality be due to inadequate control of important individual risk factors? After aggressively controlling for smoking, the estimated fine particulate pollution effect on mortality was remarkably robust. When the analysis was stratified by smoking status, the estimated pollution effect on both cardiopulmonary and lung cancer mortality was strongest for never smokers vs former or current smokers. This analysis also controlled for education, marital status, BMI, and alcohol consumption. This analysis used improved variables to control for occupational exposures and incorporated diet variables that accounted for total fat consumption, as well as for consumption of vegetables, citrus, and high-fiber grains. The mortality associations with fine particulate air pollution were largely unaffected by the inclusion of these individual risk factors in the models. The data on smoking and other individual risk factors, however, were obtained directly by questionnaire at time of enrollment and do not reflect changes that may have occurred following enrollment. The lack of risk factor follow-up data results in some misclassification of exposure, reduces the precision of control for risk factors, and constrains our ability to differentiate time dependency.

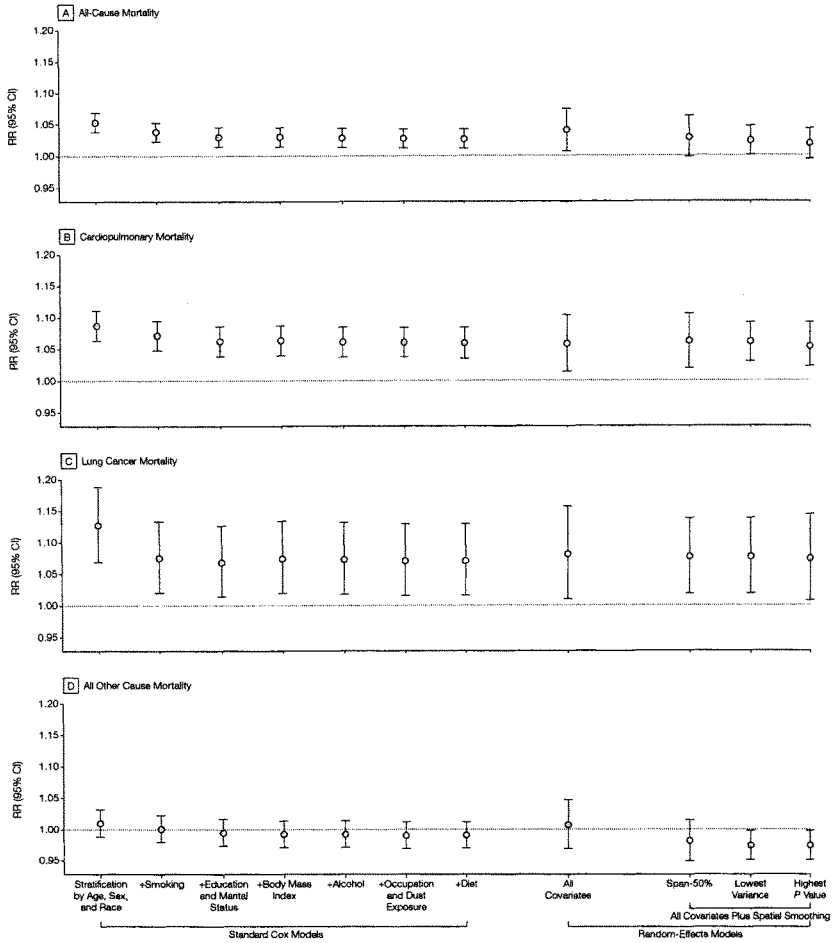
Third, are the associations between fine particulate air pollution and mortality due to regional or other spatial differences that are not adequately controlled for in the analysis? If there are unmeasured or inadequately modeled risk factors that are different across locations, then spatial clustering will occur. If this clustering is independent or random across metropolitan areas, then the spatial clustering can be modeled by adding a random-effects component to the Cox proportional hazards model as was done in our analysis. The clustering may not be independent or random across metropolitan areas due to inadequately measured or modeled

Figure 2. Nonparametric Smoothed Exposure Response Relationship



Vertical lines along x-axes indicate rug or frequency plot of mean fine particulate pollution; $PM_{2.5}$, mean fine particles measuring less than 2.5 µm in diameter; RR, relative risk; and CI, confidence interval.

Figure 3. Mortality Relative Risk (RR) Ratio Associated With 10-μg/m³ Differences of $PM_{2.5}$ Concentrations



Data presented are for 1979-1983 for the different causes of death, with various levels of controlling for individual risk factors, and using alternative modeling approaches. The 3 models with spatial smoothing allow for increasingly aggressive fitting of the spatial structure. Plus sign indicates model included previous variables (ie, smoking included stratification by age, sex, and race); $PM_{2.5}$, mean fine particles measuring less than 2.5 μm in diameter; and CI, confidence interval.

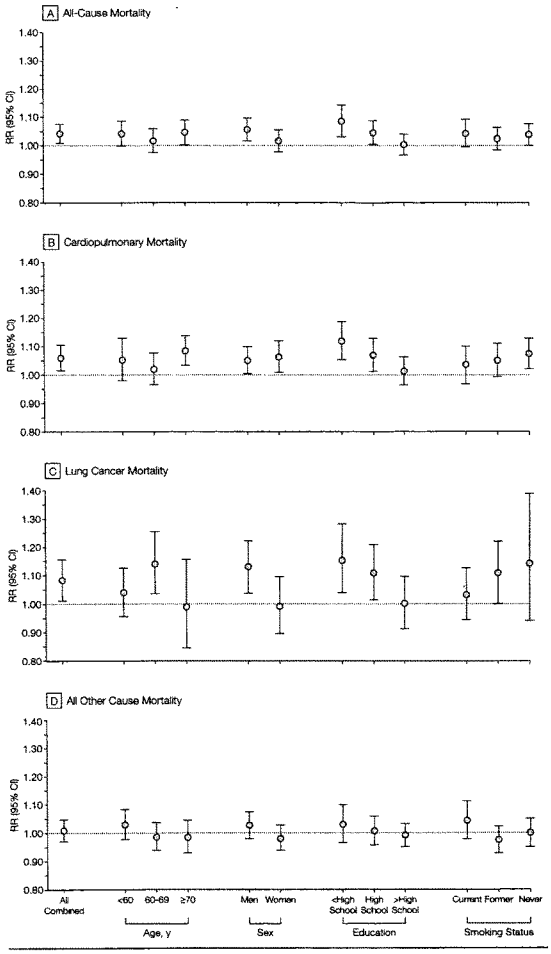
risk factors (either individual or ecological). If these inadequately measured or modeled risk factors are also spatially correlated with air pollution, then biased pollution effects estimates may occur due to confounding. However, in this analysis, significant spatial autocorrelation was not observed after controlling for fine particulate air pollution and the various individual risk factors. Furthermore, to minimize any potential confounding bias, sensitivity analyses, which directly modeled spatial trends using nonparametric smoothing techniques, were conducted. A contribution of this analysis is that it included the incorporation of both random effects and nonparametric spatial smoothing components to the Cox proportional hazards model. Even after accounting for random effects across metropolitan areas and aggressively modeling a spatial structure that accounts for regional differences, the association between fine particulate air pollution and cardiopulmonary and lung cancer mortality persists.

Fourth, is mortality associated primarily with fine particulate air pollution or is mortality also associated with other measures of particulate air pollution, such as PM_{10}, total suspended particles, or with various gaseous pollutants? Elevated mortality risks were associated primarily with measures of fine particulate and sulfur oxide pollution. Coarse particles and gaseous pollutants, except for sulfur dioxide, were generally not significantly associated with elevated mortality risk.

Fifth, what is the shape of the concentration-response function? Within the range of pollution observed in this analysis, the concentration-response function appears to be monotonic and nearly linear. However, this does not preclude a leveling off (or even steepening) at much higher levels of air pollution.

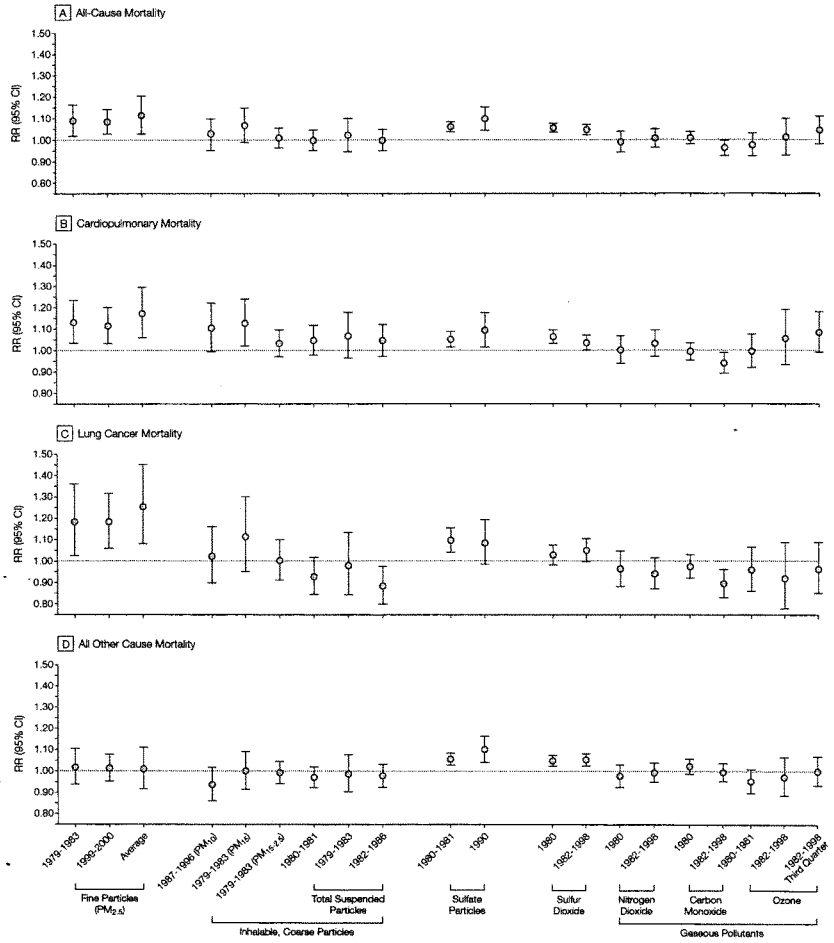
Sixth, how large is the estimated mortality effect of exposure to fine particulate air pollution relative to other risk factors? A detailed description and interpretation of the many individual risk factors that are controlled for in the analysis goes well beyond the scope of

Figure 4. Adjusted Mortality Relative Risk (RR) Ratio Associated With 10-μg/m³ Differences of $PM_{2.5}$ Concentrations



Data presented are for 1979-1983 for the different causes of death stratified by age, sex, education, and smoking status. $PM_{2.5}$ indicates mean fine particles measuring less than 2.5 μm in diameter; CI, confidence interval.

Figure 5. Adjusted Mortality Relative Risk (RR) Ratio Evaluated at Subject-Weighted Mean Concentrations



$PM_{2.5}$ indicates particles measuring less than 2.5 μm in diameter; PM_{10}, particles measuring less than 10 μm in diameter; PM_{15}, particles measuring less than 15 μm in diameter; $PM_{15-2.5}$, particles measuring between 2.5 and 15 μm in diameter; and CI, confidence interval.

this report. However, the mortality risk associated with cigarette smoking has been well documented using the CPS-II cohort.[16] The risk imposed by exposure to fine particulate air pollution is obviously much smaller than the risk of cigarette smoking. Another risk factor that has been well documented using the CPS-II cohort data is body mass as measured by BMI.[30] The Word Health Organization has categorized BMI values between 18.5-24.9 kg/m^2 as normal; 25-29.9 kg/m^2, grade 1 overweight; 30-39.9 kg/m^2, grade 2 overweight; and 40 kg/m^2 or higher, grade 3 overweight.[31] In the present analysis, BMI values and BMI values squared were included in the proportional hazards models. Consistent with previous ACS analysis,[30] BMI was significantly associated with mortality, optimal BMI was between approximately 23.5 and 24.9 kg/m^2, and the RR of mortality for different BMI values relative to the optimal were dependent on sex and smoking status. For example, the RRs associated with BMI values between 30.0 and 31.9 kg/m^2 (vs optimal) would be up to approximately 1.33 for never smokers. Based on these calculations, mortality risks associated with fine particulate air pollution at levels found in more polluted US metropolitan areas are less than those associated with substantial obesity (grade 3 overweight), but comparable with the estimated effect of being moderately overweight (grade 1 to 2).

In conclusion, the findings of this study provide the strongest evidence to date that long-term exposure to fine particulate air pollution common to many metropolitan areas is an important risk factor for cardiopulmonary mortality. In addition, the large cohort and extended follow-up have provided an unprecedented opportunity to evaluate associations between air pollution and lung cancer mortality. Elevated fine particulate air pollution exposures were associated with significant increases in lung cancer mortality. Although potential effects of other unaccounted for factors cannot be excluded with certainty, the associations between fine particulate air pollution and lung cancer mortality, as well as cardiopulmonary mortality, are observed even after controlling for cigarette smoking, BMI, diet, occupational exposure, other individual risk factors, and after controlling for regional and other spatial differences.

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Acquisition of data: Thun, Calle, Krewski, Ito, Thurston.
Analysis and interpretation of data: Pope, Burnett, Krewski, Thurston.
Drafting of the manuscript: Pope, Burnett, Ito, Thurston.
Critical revision of the manuscript for important intellectual content: Pope, Thun, Calle, Krewski, Thurston.
Statistical expertise: Pope, Burnett, Krewski.
Obtained funding: Pope, Thun, Thurston.
Administrative, technical, or material support: Pope, Calle, Krewski, Ito, Thurston.
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The Concentration–Response Relation between $PM_{2.5}$ and Daily Deaths

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Particulate air pollution at commonly occurring concentrations is associated with daily deaths. Recent attention has focused on the shape of the concentration–response curve, particularly at low doses. Several recent articles have reported that particulate matter with aerodynamic diameter ≤ 10 μm (PM_{10}) was associated with daily deaths with no evidence of a threshold. These reports have used smoothing or spline methods in individual cities and pooled the results across multiple cities to obtain estimates that are more robust. To date, fine particulate matter (aerodynamic diameter ≤ 2.5 μm; $PM_{2.5}$), a component of PM_{10}, has not been examined in this regard. We examined this association in a hierarchical model in six U.S. cities. In the first stage, we fit log-linear models including smooth functions of $PM_{2.5}$ in each city, controlling for season, weather, and day of the week. These smooth functions allowed for nonlinearities in the city-specific associations. We combined the estimated curves across cities using a hierarchical model that allows for heterogeneity. We found an essentially linear relationship down to 2 μg/m³. The same approach was applied to examine the concentration response to traffic particles, controlling for particles from other sources. Once again, the association showed no sign of a threshold. The magnitude of the association suggests that controlling fine particle pollution would result in thousands fewer early deaths per year. ***Key words:*** meta-analysis, mortality, particulate air pollution, smoothing, time series, traffic. *Environ Health Perspect* 110:1025–1029 (2002). [Online 27 August 2002] *http://ehpnet1.niehs.nih.gov/docs/2002/110p1025-1029schwartz/abstract.html*

In the last decade, a series of studies reported associations between daily concentrations of airborne particles and daily deaths (*1–3*). The magnitude of the regression coefficients in those studies indicated that particulate air pollution was associated with between 50 and 100,000 early deaths per year in the United States, and similar numbers were found in Europe. More recently, a number of large, multicity studies (*4–7*) have reported associations between airborne particles, measured in various ways, and daily deaths. The largest study demonstrated that gaseous air pollutants did not confound the association, and that none of the gaseous air pollutants showed an independent effect on daily deaths (*7*). These studies assumed a linear concentration–response relation between airborne particles and daily deaths and did not address the question of what the association looked like for particle constituents, characterized by size, physiochemical composition, or source.

In a recent study of six U.S. cities (*5*), we demonstrated that daily mortality was associated with fine particulate matter (aerodynamic diameter ≤ 2.5 μm; $PM_{2.5}$) and not with coarse particulate matter (aerodynamic diameter between 2.5 and 10 μm; $PM_{2.5-10}$). Each 10 μg/m³ increase in the 2-day mean concentration of $PM_{2.5}$ was associated with a 1.5% (95% confidence interval, 1.1–1.9%) increase in daily mortality.

Ambient $PM_{2.5}$ consists mainly of combustion particles from motor vehicles and the burning of coal, fuel oil, and wood, but also contains some crustal particles from finely pulverized road dust and soils. These sources produce particles with different characteristics, and the relative toxicity of those sources and characteristics is an area of relative recent but intense interest. In a follow-up study (*8*), we used the elemental composition of size-fractionated particles to identify several distinct source-related fractions of fine particles. We then examined the association of these fractions with daily mortality in each of the six cities and combined the city-specific results in a meta-analysis to derive overall relative risks for each fraction. We found positive associations with particles from traffic, particles from coal, and particles from residual oil combustion when included jointly in the model predicting daily deaths (*8*). The largest effect size was for residual oil particles, followed by traffic particles and then coal particles. Only the latter two associations were statistically significant, however. Again, as traditional, these analyses assumed a linear association between the various particle constituents and daily deaths.

The shape of the concentration–response relationship is critical for public health assessment, and in particular, some have speculated that thresholds might exist.

Recently, three reports have explored this question for particulate air pollution, using multicity studies in the United States. In one study, Daniels et al. (*9*) used data from 20 U.S. cities, five of which had daily measurements of PM_{10}, with the rest having measurements only one day in six. They used regression splines to model the concentration–response curve in each city and combined the results across cities. They found no evidence for a threshold. In fact, the concentration–response relation was quite linear across the entire range of exposure. In another report, Schwartz and Zanobetti (*10*) used data from 10 cities, all of which had daily measurements of PM_{10}, resulting in slightly more days of study than in the first report. They used nonparametric smoothing to model the concentration–response curve between air pollution and daily deaths in each city and combined the results across cities. Again, a linear, no-threshold relationship was seen. Schwartz and Zanobetti also performed simulations to confirm the ability of this approach to detect thresholds and other types of nonlinearity (*10*). Schwartz et al. (*11*), using data from eight Spanish cities, similarly reported a linear association between daily deaths and black smoke, an optical measure of black particles. These results held after adjusting for SO_2. To date, no similar examination of the concentration–response curve has been done for $PM_{2.5}$, or for any source components. Because $PM_{2.5}$ is now the regulated form of particulate air pollution in the United States, we here report results of such an analysis.

Materials and Methods

Air pollution data. As part of the Harvard Six Cities studies (*12*), dichotomous virtual impactor samplers were placed at a central residential monitoring site in six U.S. metropolitan areas: Boston, Massachusetts; Knoxville, Tennessee; St. Louis, Missouri; Steubenville, Ohio; Madison, Wisconsin; and Topeka, Kansas. Separate filter samples were collected of fine particles ($PM_{2.5}$) and of the coarse mass ($PM_{2.5-10}$) fraction. Integrated 24-hr samples were collected at least every other day from 1979 until the late 1980s, with daily sampling during health survey periods. For fine and coarse particle samples, mass concentration was determined separately by beta-attenuation (*13*). Except for a period

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between October 1981 and January 1984 in all cities, elemental composition of fine and coarse mass was determined by X-ray fluorescence (*14*). Elemental composition was available on 97% of these samples. In the fine fraction, 15 elements were routinely found above the limit of detection: silicon, sulfur, chlorine, potassium, calcium, vanadium, manganese, aluminum, nickel, zinc, selenium, bromine, lead, copper, and iron.

Source identification. In separate analyses for each city, we used specific rotation factor analysis to identify up to five common factors from the 15 specified elements. We specified a single element as the tracer for each factor and maximized the projection of these elements using the Procrustes rotation, a variant of the oblique rotation method (*15*). The Procrustes method allows us to use known tracers for different sources as targets for the different factors and to maximize their loadings on those factors instead of having factors defined in an entirely data-driven manner. To rescale the factor scores from the normalized scale to the mass scale (in micrograms per cubic meter), we regressed the total daily fine particle concentrations on the daily factor scores for all of the factors in separate regression models for each city and took the product of each factor score with its regression coefficient (*16*). Only sources that were significant predictors of total fine particle mass ($p < 0.10$) were considered in the mortality analyses. Further details have been published previously (*8*).

Meteorologic data. We obtained meteorologic data from the National Center for Atmospheric Research, including hourly measures of temperature, dew point temperature, and precipitation from the National Oceanographic and Atmospheric Administration weather station nearest to each city (*17*). We calculated 24-hr mean values for temperature and dew point temperature.

Mortality data. We defined the six metropolitan areas in this study as the county containing the air pollution monitor and contiguous counties (*5*). We extracted daily deaths from annual detail mortality tapes (National Center for Health Statistics) (*18*) for people who lived and died in the selected counties for the time periods with fine particulate measurements. After excluding all deaths caused by accidents and other external causes [*International Classification of Disease*, 9th Revision (*ICD-9*) (*19*), clinical modification codes 800–999], we analyzed the remaining total daily deaths.

Poisson regression of mortality. We investigated the association of daily deaths with sources of fine particles separately for each city using Poisson regression in a generalized additive model (GAM) (*20,21*). That is, in each city we assumed

$$\mathrm{Log}[E(Y_t)] = \beta_0 + \sum S_i(X_{it}), \qquad [1]$$

where Y_t is the number of deaths in the city on day t and X_{it} is the value of covariate i on day t. GAMs are distinguished by allowing us to use smooth functions S_i instead of linear terms to control for covariates, such as temperature, that may affect daily deaths in a nonlinear way. Linear functions may be used where appropriate. This approach was introduced for time series of counts in 1994 (*22*) and is now standard (*23,24*).

To control for trend and season, we used a locally weighted linear regression (LOESS) smooth function of date with a span of 0.05 (*25*). For the smooth functions of temperature and dew point temperature, we used LOESS functions with spans of 0.80. Indicator variables for day of the week also were included in the models. This is the identical model used by Schwartz et al. (*5*) and Laden et al. (*8*), and more details are provided there. To these models we added a smooth function of the mean $PM_{2.5}$ concentration on the day of death and the previous day, instead of the linear term previously used by Schwartz et al. (*5*). The smoothing window included 50% of the data, which corresponds to between four and five degrees of freedom for the air pollution relation in each city. Alternatively, we added the estimated mass for each of the source factor scores (in micrograms per cubic meter) simultaneously in the model. That is, the estimate of the mobile source factor is in a model controlling for coal-derived particles, crustal particles, and the other source factors, and vice versa. Because only the particles from traffic showed a strong linear association, and because the exposure ranges for the exposures to coal particles did not overlap sufficiently, we only used a smooth function for the traffic particles and followed Laden et al. (*8*) in treating the particle mass from the other sources as linear terms.

Hierarchical model. To combine the smooth curves across cities, we applied the approach of Schwartz and Zanobetti (*10*), as modified by Schwartz et al. (*11*). In each city, the predicted log relative risk and its pointwise standard error was computed for each 2

Table 1. Mean daily deaths in six U.S. cities and mean concentrations of $PM_{2.5}$ overall, and from the three source categories showing evidence of an association with daily deaths in Laden et al. (*8*).

City	Deaths	$PM_{2.5}$ (μg/m³)	Traffic (μg/m³)	Coal (μg/m³)	Residual oil (μg/m³)	Dates (month/year)
Boston	59	16.5	4.8	8.3	0.5	5/79–1/86
Knoxville	12	21.1	4.4	6.8	—	1/80–12/87
St. Louis	55	19.2	2.9	5.6	—	9/79–1/87
Steubenville	3	30.5	1.5	19.2	0.9	4/79–9/87
Madison	11	11.3	3.1	4.9	—	3/79–12/97
Topeka	3	12.2	2.1	7.0	—	9/79–10/88

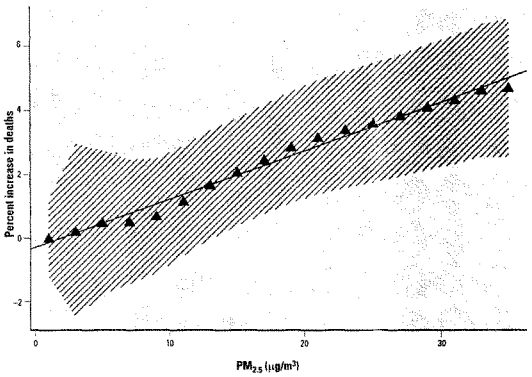

Figure 1. Overall estimated dose–response relation between total $PM_{2.5}$ and daily deaths in six U.S. cities. The estimate is obtained by combining the estimated smoothed curves in each of the cities, after controlling for weather, season, and day of the week. The shaded area indicates the pointwise 95% confidence intervals at each point. The line shown is a least-squares regression line through the estimated points.

$\mu g/m^3$ increment in exposure. These estimates are provided by the GAM function in S-plus (MathSoft, Inc., Seattle, WA). To successfully combine data across cities, we need to use a range of exposures that is common to all cities. Because high concentrations of $PM_{2.5}$ were rare, the curves were combined only in the range of 0–35 $\mu g/m^3$. The first phase of the analysis produced estimated effect sizes (log relative risks) $\hat{Y}_{ij}$ in each city i for each exposure category j. A pointwise standard error of the estimate is also estimated by GAM. To produce the combined curve, we regressed these estimates against indicator variables for each level, using inverse variance weighting and allowing for a random variance component to capture heterogeneity in the association across cities. That is, we assumed

$$\hat{Y}_{ij} \sim N\left(\beta_1 d_1 + \beta_2 d_2 + \cdots + \beta_4 d_4, V_{ij} + \delta\right), \quad [2]$$

where d_j are dummy variables for the j exposure levels, V_{ij} is the estimated variance in city i at level j, and δ is the estimated random variance component.

We used the iterative meta-regression approach of Berkey et al. (*26*) to obtain a maximum likelihood estimate of the random variance component.

The nonparametric smooth functions we use to estimate the shape of the concentration response relation use four to five degrees of freedom, and it is not clear that the source-specific relations can support so many degrees of freedom, which would entail a total of 20 degrees of freedom for all the $PM_{2.5}$ sources. In our previous report (*8*), the relation between $PM_{2.5}$ from traffic and daily deaths was estimated with considerably greater precision than for particles from other sources, most of which were not significant. Further, the range of overlap in exposures across cities was lower for coal, crustal, and residual oil factors. Therefore, in our source-specific models, we only modeled the traffic source particles using a nonparametric smooth, while controlling for $PM_{2.5}$ from the other sources using linear terms, as in Laden et al. (*8*). We then combined the estimated concentration–response relations for traffic particles similarly to what we did for $PM_{2.5}$ from the other sources.

Results

Table 1 shows the daily deaths, $PM_{2.5}$ levels, and estimated concentrations of $PM_{2.5}$ from each source. Figure 1 shows the meta-smooth dose–response relation between $PM_{2.5}$ and daily deaths in the six cities. There is no evidence of a threshold, and the relation occurs well below the U.S. Environmental Protection Agency standard of 65 $\mu g/m^3$ (*27*). The line shows the least-squares fit of a linear relation through the estimated points.

The next results come from the source component models. These models had a smooth function of $PM_{2.5}$ from traffic and linear functions of $PM_{2.5}$ from the other sources in each city. Figure 2 shows the results when we combined the estimated dose–response curves for traffic particles across the six cities. Again, there is no evidence of a threshold, and the association is essentially linear. If anything, the slope is steeper at lower concentrations. To test the robustness of the association with traffic particles to our method of controlling for particles from other sources, we re-estimated the relationship controlling for smooth functions of the estimated particle mass from other sources, rather than the linear terms. This association is shown in Figure 3 and differs little from that shown in Figure 2. We also fit linear regressions through the points shown on Figures 1 and 2. We obtained a slope of 1.5% increase in deaths per 10 $\mu g/m^3$ increase in $PM_{2.5}$ and 3% increase in deaths per 10 $\mu g/m^3$ increase in particles from traffic, which is the same as the results reported by Laden et al. (*8*). These lines are shown on the figures. This supports the assumption of a linear relationship.

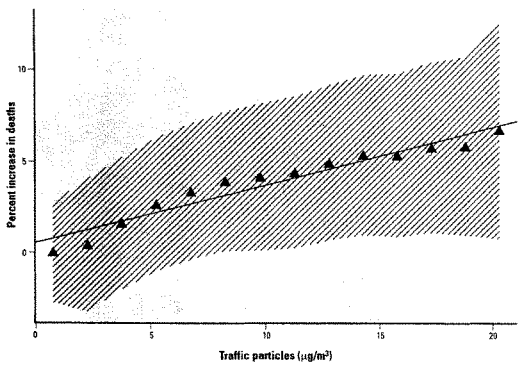

Figure 2. Overall estimated dose–response relation between $PM_{2.5}$ from traffic and daily deaths in six U.S. cities. The estimate is obtained by combining the estimated smoothed curve in each of the cities, after controlling for weather, season, and day of the week and for $PM_{2.5}$ from crustal sources, coal combustion, residual oil, salt, and metal processes as linear terms. The line shown is a least-squares regression line through the estimated points.

Discussion

We have explored the concentration–response relation between $PM_{2.5}$ and daily deaths in six U.S. cities and combined the results to obtain greater stability, while accounting for heterogeneity in response. The population mean curve shows no evidence of a threshold down to the lowest levels of $PM_{2.5}$. In fact, the curve is quite linear over the exposure range from 0 to 35 $\mu g/m^3$. This is consistent with previous results using a similar methodology but with PM_{10} (*10*) and black smoke (*11*) as the exposure metric. In addition, a different methodology, using regression splines, was applied by Daniels et al. (*9*) to PM_{10} data in different cities. They combined these spline models across 20 cities. Again, the association appeared to be quite linear without any evidence of a threshold. A spline model had previously been applied by Schwartz (*22*) to the $PM_{2.5}$ data from Boston, with a similar finding. Indeed, the original study of these data by Schwartz, Dockery, and Neas (*5*) found a significant association when limited to days below 30 $\mu g/m^3$, with a slightly larger slope. The consistency of the results on two continents, and using different techniques, suggests that this finding is robust. The concentration–response curve seen here for $PM_{2.5}$ is steeper than that previously reported (per $\mu g/m^3$) for PM_{10} (*10*). This is consistent with the previous report from this study (*5*) that coarse mass (the difference between PM_{10} and $PM_{2.5}$) is not associated with daily deaths. We note that Schwartz and Zanobetti (*10*) demonstrated in simulation studies that measurement error was not likely to distort the shape of the association. Similarly, recent studies of "harvesting" have shown that effect sizes increase rather than decrease when longer lags are taken into account; for example, high

days producing harvesting that mutes the effect on the next high day is unlikely to have distorted the shape of the association.

These results are also biologically plausible. Schwartz (*28*) pointed out that if thresholds exist in individuals, but there is a distribution of those thresholds among individuals, and if multiple genetic and predisposing illnesses each contributed to the distribution of those thresholds, then by the central limit theorem, the distribution of thresholds should approach a normal distribution. Hence, the population concentration–response curve should approach a cumulative normal curve. But the low-dose end of the cumulative normal curve is linear. To see this, consider that typical death rates in U.S. cities are 8/1,000 per year, or 2×10^{-6} per day. The normal range of variation in daily deaths in U.S. cities is a factor of two or less. Hence, the normal range of daily death probabilities in response to all risk factors is from 1 to 3×10^{-6}. Figure 4 shows the cumulative normal curve in that range of probabilities, which is quite linear. Because we are clearly in the low-dose regime, in the sense that the exposures to particles are well below the threshold for mortality for most people, this linearity is exactly what would be expected.

Figure 1 also indicates that the association reported here has public health significance. The difference between mean $PM_{2.5}$ concentrations of 10 $\mu g/m^3$ and 20 $\mu g/m^3$, which is a difference found between U.S. cities, is associated with about a 1.5% increase in deaths. In a metropolitan area of a million inhabitants, this would amount to about 130 additional early deaths per year, and in the country as a whole, these results indicate that a reduction of 10 $\mu g/m^3$ would be expected to result in about 36,000 fewer early deaths per year. Although this study does not indicate the extent to which these deaths are brought forward, other studies of the harvesting issue (*29–32*) suggest that they are considerable.

The association of daily deaths with traffic particles also has no threshold and is somewhat steeper than the association with all $PM_{2.5}$. This is consistent with the results of Laden et al. (*8*), except that they used linear terms instead of smooth functions. This study confirms that this association extends to low levels. This result has considerable public policy relevance. Recently, automotive companies have proposed using diesel engines to achieve higher fuel economy in the future. However, diesel engines produce substantially greater emissions of particles and particle precursors such as NO_x. The present results indicate that such an expansion of diesel engine use in the United States before diesel engines can meet the same particle emission levels as gasoline engines may result in important public health problems. A 1 $\mu g/m^3$ increase in the concentration of traffic particles in the United States, for example, could be associated with about 7,000 additional early deaths per year in the United States.

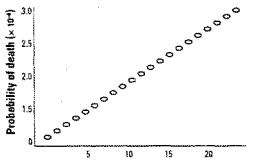

Figure 4. Cumulative normal curve versus a standardized predictor (the sum of the effects of all risk factors) over the range of exposures that correspond to daily death rates of between 1 and 3 per million, which is the observed range of variation in U.S. cities. It is quite linear in the predictor.

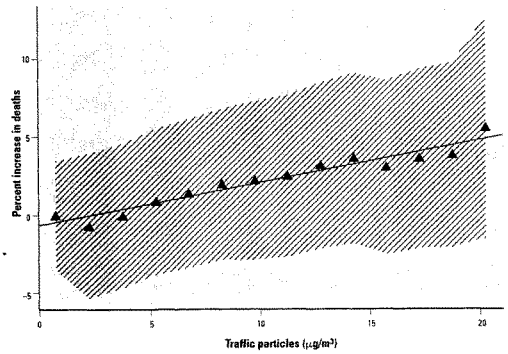

Figure 3. Overall estimated dose–response relation between $PM_{2.5}$ from traffic and daily deaths in six U.S. cities. The estimate is obtained by combining the estimated smoothed curve in each of the cities, after controlling for weather, season, and day of the week. Instead of linear terms for particles from other sources, in this analysis we controlled for smoothed terms for $PM_{2.5}$ from crustal sources, coal combustion, residual oil, and salt. The line shown is a least-squares regression line through the estimated points.

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