COMPETITIVE ENTERPRISE INSTITUTE

PARTICULATE AIR POLLUTION
WEIGHING THE RISKS

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Executive Summary

America's air quality has vastly improved in recent decades due to progressive emission reductions from industrial facilities and motor vehicles. The country achieved this success despite substantial increases in population, automobile travel, and energy production. Air pollution will continue to decline, both because more recent vehicle models start out cleaner and stay cleaner as they age than earlier ones, and also because already-adopted standards for new vehicles and existing power plants and industrial facilities come into effect in the next few years.

Nonetheless, both the Bush Administration and congressional Democrats have proposed sweeping new measures to further crack down on power plant emissions. The Administration's Clear Skies Initiative and a more stringent Democratic alternative are largely justified by claims that current levels of particulate matter (PM) pose a serious public health threat. Supporters of these bills promise substantial benefits from additional PM reductions.

Nevertheless, the benefit claims for PM reductions rest on a weak foundation. EPA based its new annual fine PM ($PM_{2.5}$) standard on a study known as the American Cancer Society (ACS) study of PM and mortality, which assessed the association between the risk of death between 1982 and 1998 with $PM_{2.5}$ levels in dozens of American cities.

Although the ACS study reported an association between PM and mortality, some odd features of the ACS results suggest that PM is not the culprit. For example, according to the ACS results, PM increased mortality in men, but not women; in those with no more than a high school degree, but not those with at least some college education; in former-smokers, but not current- or never-smokers; and in those who said they were moderately active, but not those who said they were very active or sedentary.

These odd variations in the relationship between $PM_{2.5}$ and mortality seem biologically implausible. Even more surprising, the ACS study reported that higher $PM_{2.5}$ levels were *not* associated with an increased risk of mortality due to respiratory disease; a surprising finding, given that PM would be expected to exert its effects through the respiratory system.

EPA also ignored the results of another epidemiologic study that found no effect of $PM_{2.5}$ on mortality in a cohort of veterans with high blood pressure, even though this relatively unhealthy cohort should have been more susceptible to the effects of pollution than the general population. The evidence therefore suggests that the existing annual standard for $PM_{2.5}$ is unnecessarily stringent. Attaining the standard will be expensive, but is unlikely to improve public health.

EPA also promulgated a standard for daily $PM_{2.5}$ levels. Hardly any areas exceed this standard, making it moot for policy purposes. Nevertheless, the epidemiology of short-term PM exposure and mortality suffers from deficiencies that call into question the extent to which typical short-term increases in PM levels can increase mortality.

Sulfate PM—the type of PM caused by coal power plant emissions—is a particularly implausible culprit as a cause of increased mortality. Ammonium sulfate, the main form of sulfate PM, is used as an inactive control substance in human studies assessing the

health effects of inhaling acidic aerosols. Inhaled magnesium sulfate is used therapeutically to *reduce* airway constriction in asthmatics. Sulfate is also naturally present in bodily fluids at levels many times the amount that could be inhaled from air pollution.

The evidence suggests that exposure to PM at current levels likely has little or no effect on mortality in most of the United States. Regardless, processes already set in motion guarantee substantial PM reductions in coming years. Additional near-term reductions in PM are probably best achieved by dealing with the stock of high-polluting older vehicles that account for a substantial portion of ambient PM levels in metropolitan areas. This flexible, more cost-effective approach is far more likely to result in net public health benefits than other proposals that are the focus of current legislative and regulatory activity and debate.

Introduction

There is no question that high levels of air pollution can kill. About 4,000 Londoners died during the infamous five-day "London Fog" episode of December 1952, when soot and sulfur dioxide soared to levels tens of times greater than the highest levels experienced in developed countries today, and visibility dropped to less than 20 feet.[1] A number of other high-pollution episodes up through the 1970s exacted a similarly horrifying toll.[2]

Fortunately, the United States has been very successful in reducing air pollution. Due to a combination of technological advances and regulatory intervention, pollution levels have been declining for decades, despite large increases in population, energy use, and driving.

Nevertheless, many health researchers, regulators, and environmental activists are concerned that airborne particulate matter (PM), especially smaller particulates known as PM_{10} and $PM_{2.5}$,[3] might still be causing tens of thousands of premature deaths each year, even at the relatively low levels currently found in most areas of the United States.[4] Policymakers and environmental activists have recently focused special attention on the health effects of power-plant emissions, which are a significant contributor to $PM_{2.5}$ levels in parts of the eastern United States.

Bills introduced by Senator James Jeffords (I-VT) and the Bush Administration would require cuts in power plant emissions well beyond current requirements; advocates for both proposals claim they would save thousands of lives per year.[5] Environmental

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[1] I. M. Goklany, *Clearing the Air: The Real Story of the War on Air Pollution* (Washington, DC: Cato, 1999).

[2] Ibid.

[3] PM_{10} and $PM_{2.5}$ refer, respectively, to airborne particulates less than or equal to 10 or 2.5 micrometers in diameter.

[4] R. Wilson and J. Spengler, eds., *Particles in Our Air: Concentrations and Health Effects* (Cambridge, MA: Harvard University Press, 1996).

[5] Senator Jeffords's bill S.366 is known as the "Clean Power Act," while the Bush Administration's proposed "Clear Skies Initiative" is embodied in S.485 and H.R.999. The Jeffords bill would require substantial cuts in sulfur dioxide (SO_2), nitrogen oxides (NOx), mercury, and carbon dioxide by 2008 (see table below). The Clear Skies Initiative does not address carbon dioxide emissions, and cuts other emissions by slightly less than the Jeffords bill on a schedule extending out to 2018.

Comparison of Power Plant Emissions under the Jeffords and Bush Proposals*

Pollutant	Estimate for 2000	Clean Power Act	Clear Skies Initiative
SO_2	11.2	2.25	3.00
NOx	5.1	1.51	1.70
Mercury	48	5	15

* SO_2 and NOx emissions are in millions of tons per year. Mercury emissions are in tons per year. The Clean Power Act caps would take effect in 2008, while the Clear Skies Initiative caps would take effect in 2018. Clear Skies also includes intermediate caps for SO_2 and NOx of, respectively,

groups have published a series of reports claiming substantial harm to public health from power plant emissions.[6] These groups ardently oppose the Clear Skies Initiative as well as the Bush Administration's proposed reform of the Clean Air Act's New Source Review regulation, arguing that it would allow substantial increases in power plant emissions.[7]

PM health effects studies have reported both acute increases in death and disease due to daily variation in PM levels, as well as increases in death due to chronic exposure to elevated PM levels. The Environmental Protection Agency (EPA) promulgated annual-average and daily PM_{10} health standards in 1987. However, after reviewing recent PM health research, EPA in 1997 decided to also promulgate health standards for $PM_{2.5}$ specifically.

The annual-average $PM_{2.5}$ standard is controversial because it is among the most stringent ever promulgated by EPA, and will be difficult and expensive to attain in many areas that do not currently comply with it. EPA and environmental activists believe attaining the $PM_{2.5}$ standard will save as many as tens of thousands of lives per year and mitigate respiratory symptoms for hundreds of thousands of people.[8]

On the other hand, critics of EPA's interpretation of the PM health literature contend that the effects of low-level PM exposure are probably much smaller than advocates of $PM_{2.5}$ regulation have concluded. The effects of high-pollution episodes such as the London Fog were obvious, even without epidemiologic analysis, because both pollution levels and mortality soared by many times above typical levels. However, current PM levels at worst increase mortality and disease by a few percent above background rates. Such small relative changes can't be observed directly and must be teased out using the statistical analysis methods of epidemiology.

However, epidemiological analyses are susceptible to various methodological biases and errors that could cause misattribution of health effects to PM when they are caused by another pollutant or by factors unrelated to pollution, such as weather or diet. Some epidemiologists believe that epidemiologic methods are not even capable of accurately teasing out very small increases in health risks. Although epidemiologic studies have had mixed results on the link between particulates and health, the media and politicians have

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4.5 million and 2.1 million tons that take effect in 2008, and a 26-ton-per-year cap for mercury that would take effect in 2010.

[6] See, for example, Clean Air Task Force, "Power to Kill: Death and Disease from Power Plants Charged with Violating the Clean Air Act" (Boston: 2001); Public Interest Research Group, "Darkening Skies: Trends toward Increasing Power Plant Emissions" (Washington, DC: 2002); and Clean Air Task Force, "Death, Disease and Dirty Power: Mortality and Health Damage Due to Air Pollution from Power Plants" (Boston: 2000).

[7] See, for example, Public Interest Research Group, "Bush Policies would make Air Smoggier," July 1, 2002, www.commondreams.org/news2002/0701-05.htm. New Source Review is the regulatory regime for new and modified industrial sources of pollution.

[8] See, for example, Abt Associates, "The Particulate-Related Health Benefits of Reducing Power Plant Emissions" (Bethesda, Maryland: 2000); Clean Air Task Force, "Death, Disease and Dirty Power."

often failed to convey the nuances, uncertainties, and controversies surrounding the science of PM health effects.[9]

Critics of EPA's PM standards and the pending power plant-related bills also contend that the costs of meeting the annual $PM_{2.5}$ standard would exceed the value of the health benefits achieved, resulting in a net loss in the public's welfare.

Overview of this Report

This study assesses current PM health risks and identifies PM air pollution policies that are most likely to generate net public health benefits. To that end, it sets up the policy discussion with analyses of baseline air pollution levels and trends, the weight of the evidence on PM health effects at current ambient levels, and likely costs and benefits of attaining current air pollution standards. The final section draws on these discussions to recommend policies geared toward maximizing net benefits to society.

Air pollution sources and trends. Appropriate policy depends not only on current pollution levels, but also on expected future pollution levels. This paper begins with a summary of air pollution trends, current levels, and prospects, based on pre-existing trends and regulations already on the books. It shows that PM and other kinds of air pollution have been declining for decades—few areas of the United States now have high air pollution levels, relative either to current health standards or past levels. The study concludes that baseline trends—mainly turnover of the vehicle fleet—combined with existing requirements for industrial sources, will result in large reductions in all major air pollutants in coming years. This means that air pollution has been largely addressed as a long-term problem, but also that these already-adopted measures will take time to come to fruition.

PM health effects. The report then focuses on the state of the science for both long-term and short-term health effects of PM at current levels. Health-effects studies have reported associations between elevated PM and increases in both death and disease. I focus on mortality, because this is by far the most serious adverse effect attributed to PM, and because there is widespread agreement that the vast majority of the benefits from PM reductions would result from reductions in premature death.[10] Furthermore, the discussion of the strength of the evidence on PM and premature death applies equally well to PM and increased disease, because the same suite of statistical methods is used for both types of health studies.

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[9] See, for example, C. Seabrook, "Dirty Air Raises Cancer Risk, Study also Links Pollution to Heart Attacks," *Atlanta Journal Constitution*, March 6, 2002; E. Pianin, "Study Ties Pollution, Risk of Lung Cancer; Effects Similar to Secondhand Smoke," *Washington Post*, March 6, 2002; and U. S. Senate, Committee on Environment and Public Works, "Majority Report on the Clean Power Act of 2002," June 27, 2002.

[10] For example, a study commissioned by a coalition of environmental groups estimates that 95 percent of the benefits of PM reductions would come from reductions in mortality, while EPA predicts more than 90 percent of benefits would come from mortality reductions (Abt Associates, "The Particulate-Related Health Benefits of Reducing Power Plant Emissions," and EPA, *Technical Addendum: Methodologies for the Benefit Analysis of the Clear Skies Initiative* (Washington, DC, 2002), www.epa.gov/clearskies/tech_adden.pdf).

The report concludes that current PM levels are generally too low to increase risk of death due to long-term exposure and that EPA's current annual-average $PM_{2.5}$ standard is more stringent than necessary to protect public health. The weight of the evidence for short-term health effects is less clear. Although many studies have reported increases in death and disease due to daily increases in PM levels, a number of researchers have raised substantive concerns over whether PM is the pollutant responsible for the observed health effects, whether pollution reduces life-expectancy by more than a few days, whether there is a threshold level below which PM has no health effects, and whether the confounding effects of non-pollution factors such as weather have been adequately addressed. Recently discovered software glitches may also have caused dozens of studies to overestimate the acute health effects of PM.

A detailed review of the dozens of studies of short-term PM health effects is beyond the scope of this report, which aims to give the reader an understanding of the key issues and the current state of the science. The report concludes that there is still substantial uncertainty in the degree of increased mortality due to daily variation in PM levels, though the evidence suggests that PM is at worst shortening life by no more than a few days in already-frail individuals. In addition, progressive refinements in the research literature have tended to reduce the size of the estimated effects. It also concludes that the issue is currently moot for policy purposes, since no more than a few percent of monitoring locations exceed the federal health standard for daily PM_{10} or $PM_{2.5}$ levels.

Net benefits for public health. People ultimately bear regulatory costs through reductions in their disposable income, because regulations increase the costs of producing useful goods and services. People, on average, use their income to increase health and safety for themselves and their loved ones. Therefore reducing people's income reduces their health. Only by ensuring that a given policy will do more good than harm can policymakers ensure *net* benefits for public health and welfare. Because of the high projected costs of attaining the current annual $PM_{2.5}$ standard and the small health benefits that would accrue, requiring attainment of the standard on the current regulatory timeline would likely cause net harm to public health.

Policy considerations. The first three sections of the report feed into an assessment of policy options, including the following conclusions:

- Based on the weak evidence for long-term health effects of $PM_{2.5}$ at levels below 20 $\mu g/m^3$, EPA could relax the annual $PM_{2.5}$ standard from 15 $\mu g/m^3$ to 20 $\mu g/m^3$ while still adequately protecting public health, and avoiding most of the costs of attaining the current standard.
- Because PM air pollution has been mitigated as a *long-term* problem, policy should focus on *near-term* measures to mitigate PM in areas that still have high levels.
- Most motor-vehicle pollution comes from a small percentage of older vehicles. Incentives to retrofit or scrap these vehicles would generate large near-term PM reductions at relatively low cost compared to other proposals currently on the table, such as the Bush Administration's Clear Skies Initiative and Senator Jeffords' Clean Power Act.

Pollution Levels, Sources, and Trends[11]

Ambient air pollution levels have been declining almost everywhere in the United States for decades. Average levels of carbon monoxide (CO) and sulfur dioxide (SO_2) declined 75 percent during the last 30 to 40 years, while nitrogen oxides (NOx) declined more than 40 percent.[12] Virtually all areas of the country now comply with federal health standards for these pollutants.[13] Eighty-seven percent of monitoring locations now comply with the federal one-hour ozone standard, up from 50 percent in the early 1980s. Only 60 percent comply with EPA's new, more stringent ozone standard, known as the "eight-hour standard." However, most eight-hour ozone non-attainment locations are relatively close to the standard, with 70 percent exceeding the standard by 10 percent or less.[14]

Particulate matter has also declined substantially. A number of local agencies collected data on PM levels as far back as the early 1900s, while national data go back as far as the 1950s.[15] These early PM measurements focused on "dustfall," "smoke density," and total suspended particulates (TSP; that is, all particulates suspended in air) until 1988, when EPA began requiring states to collect data on PM_{10}.

Data from the early 1900s through the 1960s and 1970s show that dustfall and TSP declined throughout the 20th Century. For example, dustfall in Pittsburgh declined by about 90 percent between the early 1900s and 1977, while TSP levels declined about 60 percent between the late 1950s and 1975. Smoke density in Chicago declined by 50 percent between 1911 and 1933. Cincinnati achieved a 50 percent decline in dustfall between the 1930s and 1960s. Many other U.S. metropolitan areas also achieved substantial PM declines.[16]

TSP data from dozens and later hundreds of locations around the U. S. are available from 1957 to the early 1990s. These data show average TSP levels in urban and suburban areas declined by roughly 50 percent during this period. Rural particulate levels actually increased about 80 percent from 1957 to 1970, though rural levels started out at one-fourth to one-sixth of levels in populated areas.[17]

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[11] For a more detailed discussion and analysis of air pollution trends, see Joel Schwartz, "Understanding Air Pollution: Trends, Health Effects, and Current Issues" (Washington, DC: Cato, May 2003, forthcoming).

[12] Goklany, *Clearing the Air*, F. W. Lipfert and S. C. Morris, "Temporal and Spatial Relations between Age Specific Mortality and Ambient Air Quality in the United States: Regression Results for Counties, 1960-97," *Occupational and Environmental Medicine*, vol. 59, no. 3 (2002), pp. 156-74.

[13] Three of 557 monitoring locations exceed the CO health standard. Two of 667 monitoring locations exceed the SO_2 standard. The entire country attains the NOx standard. (Based on analysis of AirData pollution monitoring data reports downloaded from EPA, www.epa.gov/aqspubl1/select.html.)

[14] Based on analysis of ozone monitoring data for 1982 through 2002 downloaded from www.epa.gov/aqspubl1/select.html.

[15] Goklany, *Clearing the Air*, and references therein.

[16] See figures 1-2 and 1-7 in Goklany, *Clearing the Air* for graphical displays of early PM trends in several cities as well as citations for the original data sources.

[17] See figure 3-1 in Goklany, *Clearing the Air*.

PM_{10} data are now collected at hundreds of unique locations around the U.S. Data for many sites go back to 1988. EPA has two health standards for PM_{10}—a daily standard of 150 micrograms per cubic meter ($\mu g/m^3$) and an annual-average standard of 50 $\mu g/m^3$.[18] PM_{10} levels declined 19 percent from 1991 to 2000 and more than 96 percent of PM_{10} monitoring locations now meet all federal PM_{10} health standards.[19] There is also evidence of large declines from major sources of PM emissions. For example, PM emissions from diesel trucks declined 83 percent between 1975 and 2000.[20] As noted earlier, SO_2 emissions, some of which are converted to sulfate PM, have also declined substantially.

Based on evidence that very fine particulates might be the most problematic for health, EPA promulgated new PM standards in 1997, this time for $PM_{2.5}$.[21] More than 97 percent of monitoring locations comply with the daily $PM_{2.5}$ standard. However, only 70 percent comply with the annual standard. After the eight-hour ozone standard, the annual $PM_{2.5}$ standard is EPA's most stringent.

Although EPA has required nationwide $PM_{2.5}$ data collection only since 1999, $PM_{2.5}$ data were also collected from 1979 to 1983 in 51 large metropolitan areas. Based on these data, annual-average $PM_{2.5}$ levels have declined about 33 percent during the last 20

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[18] The annual standard requires that mean annual PM_{10} level, averaged over the last three years, be less than or equal to 50 micrograms per cubic meter ($\mu g/m^3$) at each monitoring location in a given region. Until recently, the daily standard required that during a 24-hour averaging period, PM_{10} levels could not exceed 150 $\mu g/m^3$ on more than 3 days in any consecutive three-year period. EPA revised the standard in 1997 as follows: For each of the last three years, determine the daily PM_{10} reading that represents the 99th percentile for the year, and average these three readings. A region exceeds the standard if the result is greater than 150 $\mu g/m^3$ for at least one monitoring location in the region. (EPA, "National Ambient Air Quality Standards for Particulate Matter: Final Rule," *Federal Register*, July 18, 1997, pp. 38652-753).

[19] Based on analysis of AirData pollution monitoring data reports downloaded from EPA, www.epa.gov/aqspubl1/select.html.

[20] Alan W. Gertler et al., "Emissions from Diesel and Gasoline Engines Measured in Highway Tunnels," Health Effects Institute, January 2002, www.healtheffects.org/Pubs/GertGros.pdf. The 83 percent figure represents a decrease in emissions per mile of travel. According to the federal Bureau of Transportation Statistics, total diesel truck mileage increased 180 percent from 1975 to 1999, so the decrease in *total* truck PM_{10} emissions is about 52 percent (calculate this as follows: set total truck PM emissions in 1975 equal to an arbitrary baseline level of one, then multiply by an 83 percent decrease in the emission rate, and then by a 180 percent increase in total mileage: 1 * (1 - 0.83) * (1 + 1.8) = 0.48, or a 52 percent reduction from the initial level). There are no data on ambient diesel PM levels over time in American cities, and these estimates of changes in total emissions and the emissions rate for diesel PM can't easily be used to infer percent changes in ambient levels. Ambient levels are probably more closely related to diesel PM emissions per unit of land area. Because American metropolitan areas have generally become less densely populated during the last 25 years, the reduction in emissions per unit of land area is probably closer to or even greater than the 83 percent figure. (Truck mileage data come from Bureau of Transportation Statistics, "National Transportation Statistics, 2001," publication BTS02-06, www.bts.gov/publications/nts/index.html, Table 1-29).

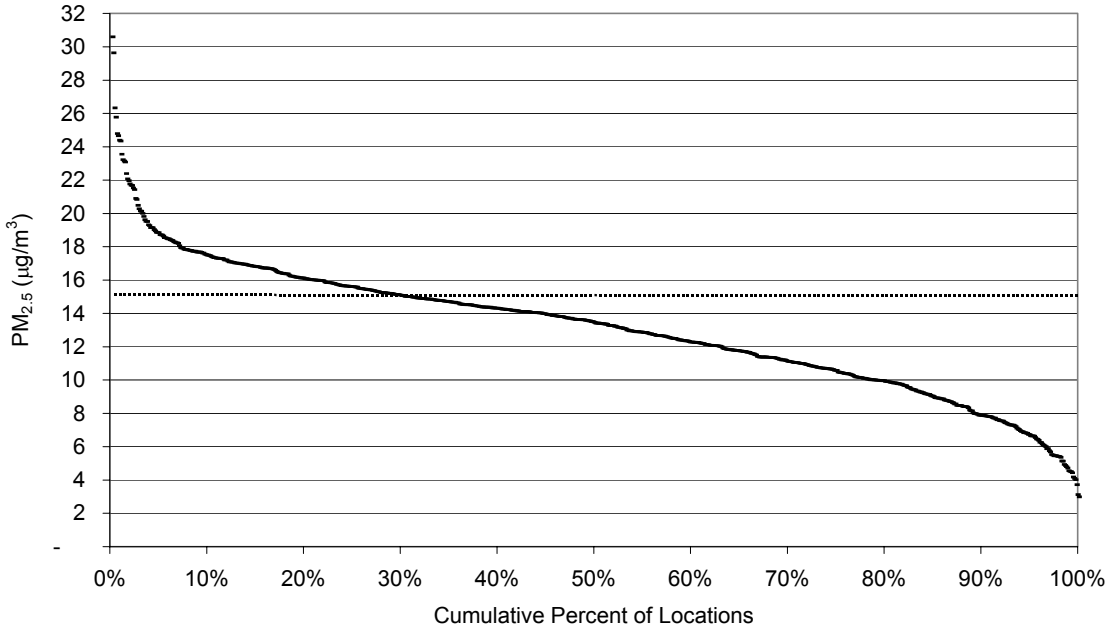
[21] The annual $PM_{2.5}$ standard requires that the mean annual particulate level, averaged over the last three years, be less than or equal to 15 $\mu g/m^3$ for each monitoring location in a given region. Attainment of the daily standard is calculated as follows: For each of the last three years, determine the daily $PM_{2.5}$ reading that represents the 98th percentile for the year, and average these three readings. A region exceeds the standard if the result is greater than 65 $\mu g/m^3$ for at least one monitoring location in the region. (EPA, "National Ambient Air Quality Standards for Particulate Matter: Final Rule.")

years.[22] These declines occurred across the board, with the worst areas achieving the largest reductions.[23]

Figure 1 shows the distribution of annual-average $PM_{2.5}$ levels for all U.S. monitoring locations. The dotted line marks the 15 $\mu g/m^3$ federal health standard. The graph shows that most $PM_{2.5}$ non-attainment locations have $PM_{2.5}$ levels relatively close to the standard—three-quarters of non-attainment locations exceed the standard by less than 20 percent. Seventeen of the worst 20 locations (with $PM_{2.5}$ ranging from 21.4 to 30.6 $\mu g/m^3$) are in California, specifically the southern portion of the Central Valley, parts of Los Angeles, and the greater San Bernardino area.[24]

Figure 1

Distribution of Annual-Average $PM_{2.5}$ Levels for All U.S. Monitoring Locations, 1999-2001



The graph plots the annual-average of $PM_{2.5}$ readings for 1999-2001 at 839 locations across the United States (all locations with three years of data), ranked from worst to best. The dotted line marks EPA's 15 $\mu g/m^3$ annual $PM_{2.5}$ standard.

Figure 2 displays the distribution of high daily $PM_{2.5}$ levels across the U.S. The graph plots the average of the 99th percentile of daily $PM_{2.5}$ levels for 1999-2001, and includes

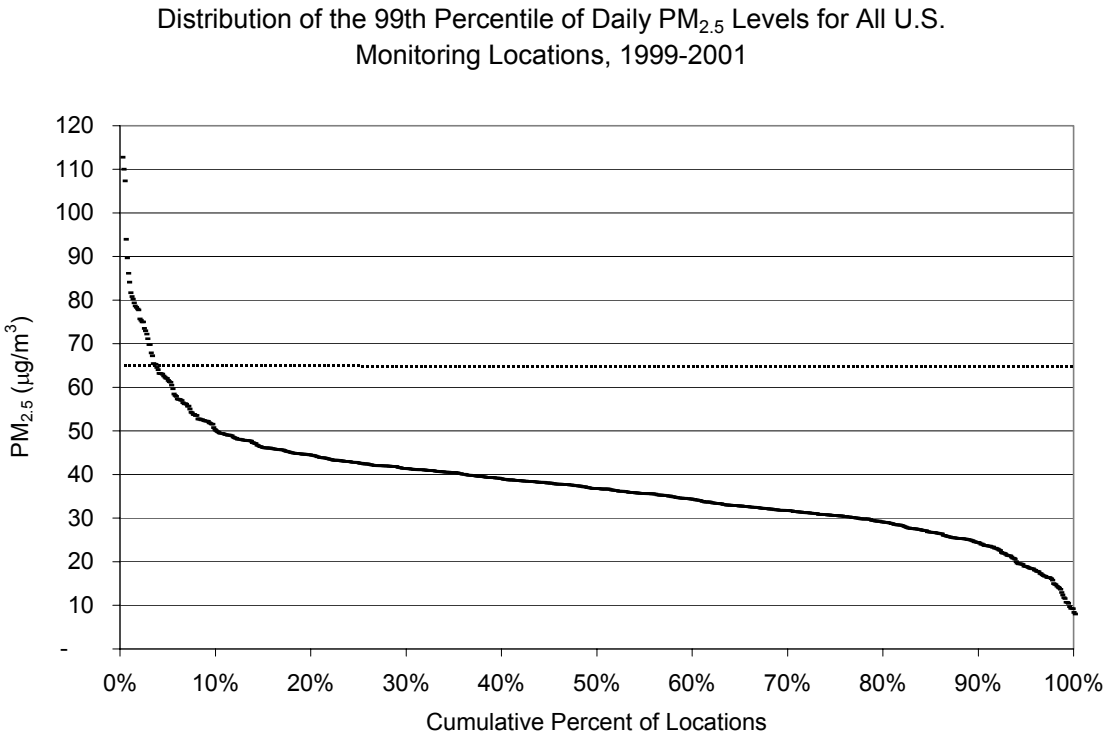
[22] C. A. Pope, 3rd et al., "Lung Cancer, Cardiopulmonary Mortality, and Long-Term Exposure to Fine Particulate Air Pollution," *Journal of the American Medical Association*, vol. 287, no. 9 (2002), pp. 1132-41.

[23] Ibid.

[24] The other three locations are in Atlanta, GA, Birmingham, AL, and a rural area of Sumner County, TN. The top 11 locations, ranging from 23.1 to 30.6 $\mu g/m^3$, are all in California.

all locations with three years of data (a total of 839 locations). As the graph shows, only a few areas of the country ever have high daily $PM_{2.5}$ levels.[25] Among the 30 locations with values greater than 65 $\mu g/m^3$, 26 are in California, including the top 16.[26] Thus, as for annual-average $PM_{2.5}$, few areas have very high levels.

Figure 2



The graph plots the average of the 99th percentile of daily $PM_{2.5}$ readings for 1999-2001 at 839 locations across the United States (all locations with three years of data), ranked from worst to best. The dotted line marks EPA's 65 $\mu g/m^3$ daily $PM_{2.5}$ benchmark. But note that the federal standard is based on the 98th percentile of daily $PM_{2.5}$ values, rather than the 99th percentile. This chart therefore overestimates the number of locations that exceed the daily $PM_{2.5}$ standard.

PM Composition and Sources

Particulate matter can be emitted directly into the air as "primary particulates," or formed from gaseous "precursors"—NOx, SO_2 and volatile organic compounds (VOCs)—through chemical transformations in the atmosphere, resulting in "secondary particulates." As a result, determining the sources of PM in air requires sophisticated

[25] 3.4 percent of monitoring locations have 99th percentile daily $PM_{2.5}$ levels exceeding 65 $\mu g/m^3$. This is greater than the percent of locations that actually exceed the federal daily $PM_{2.5}$ standard. The federal standard is based on the 98th percentile of daily $PM_{2.5}$ readings. However, the EPA online database of pollution monitoring data provides only the 99th percentile of daily readings.

[26] The other four are Pocatello, ID, Liberty, PA, Hammond, IA, and Columbus, GA.

"source apportionment" studies that combine measurements of PM composition in air with profiles of the composition of emissions from various sources of primary and secondary PM, such as gasoline and diesel vehicles, power plants and factories, and soils or other geological materials. These studies show that PM sources and composition vary by location and season. A number of generalizations can be made, as follows:[27]

Sulfate, secondary PM derived from gaseous SO_2, makes up a larger portion of $PM_{2.5}$ in the east than the west, due mainly to much greater use of coal for electricity in the east.[28] Based on recent studies, sulfate averages about 25 percent of $PM_{2.5}$ mass in the northeast, 30 percent in the southeast, and more than 40 percent in Washington, DC and Virginia.[29] Daily fluctuations can result in substantial variation around these long-term averages.[30] EPA estimates that about two-thirds of sulfate-forming SO_2 emissions come from coal-fired power plants. Sulfate accounts for a much smaller portion of $PM_{2.5}$ in the west, for example, a few percent in Denver, several percent in California's Central Valley, and about nine to 17 percent in southern California.[31]

Organic and elemental carbon (OC and EC), mainly from cars and trucks, but also due to agricultural burning, residential wood burning, and meat cooking, make up a large portion of $PM_{2.5}$ in the west and in urban areas almost everywhere.[32] The organic carbon includes both primary and secondary particulates. Based on the studies referenced above, EC and OC together typically make up about 20 to 60 percent of $PM_{2.5}$ mass.

These same studies show nitrates, secondary particulates derived from NOx emissions, are a small contributor to $PM_{2.5}$ in the east, but generally make up 15 to 40 percent of $PM_{2.5}$ in western areas.[33] Cars and trucks are the overwhelming sources of NOx in the west, with power plants contributing about 10 to 15 percent.[34]

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[27] This discussion presents mainly averages over periods of weeks to months. But particulate composition can fluctuate from day to day and by season, based on variations in emissions levels and meteorological factors, such as winds, temperature, and sunlight.

[28] Sulfate is typically in the form of ammonium sulfate, formed by reaction with ammonia in the atmosphere.

[29] Mei Zheng et al., "Source apportionment of $PM_{2.5}$ in the Southeastern United States Using Solvent-Extractable Organic Compounds as Tracers," *Environmental Science and Technology*, vol. 36 (2002), pp. 2361-71, Glen R. Cass et al., "Determination of Fine Particle and Coarse Particle Concentrations and Chemical Composition in the Northeastern United States, 1995," prepared for NESCAUM, December 1999.

[30] See, for example, William K. Modey et al., "Fine particulate ($PM_{2.5}$) Composition in Atlanta, USA: Assessment of the Particle Concentrator-Brigham Young University Organic Sampling System, PC-BOSS, During the EPA Supersite Study," *Atmospheric Environment*, vol. 35 (2001), pp. 6493-6502.

[31] Bong Mann Kim, Solomon Teffers, and Melvin D. Zeldin, "Characterization of $PM_{2.5}$ and PM_{10} in the South Coast Air Basin of Southern California: Part 1—Spatial Variations," *Journal of the Air and Waste Management Association*, vol. 50 (2000), pp. 2034-44, John G. Watson et al., "Receptor Modeling Application Framework for Particle Source Apportionment," submitted to *Chemosphere*, Judith C. Chow and John G. Watson, "Review of $PM_{2.5}$ and PM_{10} Apportionment for Fossil Fuel Combustion and other Sources by the Chemical Mass Balance Receptor Model," *Energy and Fuels*, vol. 16 (2002), pp. 222-60.

[32] Residential wood combustion is of course a more important source in winter than in other seasons.

[33] As with sulfate, most nitrate is in the form of ammonium nitrate.

[34] According to the California Air Resources Board (CARB), power plants contribute only two percent of total NOx emissions in southern California and in California's Central Valley. California generates

For PM_{10}, geological material—that is, soil and dust—typically makes up 15 to 50 percent of total mass, with some combination of OC, sulfates, and nitrates accounting for most of the rest.[35] Both PM_{10} and $PM_{2.5}$ also generally contain trace amounts of various metals, such as iron, vanadium, selenium, and zinc.

Future PM Levels

Pollution will continue to decline even without any additional regulatory intervention. Motor vehicles are generally the largest source of $PM_{2.5}$-forming pollution in populated areas. But emissions from gasoline vehicles are declining by about six to 12 percent per year, as lower-emitting and more durable newer models replace older high-polluters.[36] Likewise, EPA projects diesel truck NOx emissions are declining by about five percent per year and PM emissions by about three percent per year due to fleet turnover.[37] EPA projects regulations that will take effect between 2004 and 2009 will reduce emissions from new cars and trucks by an additional 80 to 90 percent below current new-vehicle requirements.[38] Based on these trends and the upcoming regulations, per-mile emissions from gasoline vehicles will decline about 90 percent during the next 20 years, while the current fleet-turnover trend, combined with future new-truck requirements will reduce diesel PM by 75 percent and NOx by 80 percent.[39]

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hardly any electricity from coal. (NOx emissions for the western U.S. were downloaded from EPA's National Emissions Inventory (NEI) database, www.epa.gov/air/data/repsst.html. Regional emission inventories for California were downloaded from CARB's web site, www.arb.ca.gov/emisinv/maps/statemap/abmap.htm).

[35] Kim et al., "Characterization of $PM_{2.5}$ and PM_{10} in the South Coast Air Basin of Southern California: Part 1—Spatial Variations;" Cass et al., "Determination of Fine Particle and Coarse Particle Concentrations and Chemical Composition in the Northeastern United States, 1995." Geological material and road dust can make up more than 70 percent of PM_{10} in a few cases, such as Calexico, CA and Las Vegas (Chow and Watson, "Review of $PM_{2.5}$ and PM_{10} Apportionment for Fossil Fuel Combustion").

[36] The data showing this come mainly from on-road remote sensing, vehicle inspection programs, and tunnel studies of vehicle emissions (see Joel Schwartz, "No Way Back: Why Air Pollution Will Continue to Decline" (Washington, DC: American Enterprise Institute, April 2003), and A. J. Kean et al., "Trends in Exhaust Emissions from In-Use California Light-Duty Vehicles, 1994-2001" (Warrendale, Pennsylvania: Society of Automotive Engineers, 2002)).

[37] EPA, "Regulatory Impact Analysis: Heavy-Duty Engine and Vehicle Standards and Highway Diesel Fuel Sulfur Control Requirements" (Washington, DC: 2000).

[38] Ibid., EPA, "Regulatory Impact Analysis: Tier 2 / Gasoline Sulfur Final Rulemaking" (Washington, DC: 1999).

[39] Schwartz, "No Way Back," and EPA, "Regulatory Impact Analysis: Tier 2 / Gasoline Sulfur Final Rulemaking." Increases in vehicle travel will offset only a small percentage of these pollution reductions. For example, if per-mile emissions decline by 85 percent and total vehicle miles traveled increase by 40 percent, total emissions would decline by 78 percent (calculate this as follows: set current emissions equal to an arbitrary baseline level of one, then multiply by an 85 percent decrease in the emission rate, and then by a 40 percent increase in total mileage, as follows: 1 * (1 - 0.85) * (1 + 0.4) = 0.22, or a 78 percent reduction from the initial level). Measurements of recent trends in vehicle emissions confirm this. For example, Kean et al. found that between 1994 and 2001, total HC and NOx emissions from gasoline vehicles in the San Francisco Bay Area declined 63 percent and 43 percent, respectively, even though gasoline consumption increased 13 percent, and SUVs and light trucks increased from 31 percent to 38 percent of the vehicle fleet (Kean et al., "Trends in Exhaust Emissions from in-Use California Light-Duty Vehicles, 1994-2001").

Industrial emissions will also continue to decline due to already-adopted regulations. For example, starting in 2004, EPA will cap warm-season NOx emissions from coal-fired power plants and industrial boilers at 60 percent below current levels, while power-plant SO_2 emissions will be capped at 20 percent below 2000 levels, and 43 percent below 1990 levels, by 2010.[40]

These results suggest that natural fleet turnover, along with already adopted regulations, will remove most remaining air pollutant emissions during the next two decades.

Epidemiologic Basis for PM Health Concerns

Concerns about the health effects of PM rest on the results of epidemiologic studies that have found associations between ambient PM levels and increases in death and disease. The gold standard for epidemiologic studies is the randomized, controlled experiment, in which participants are randomly assigned to "treatment" and "control" groups. This technique is used in the final stages of drug development to ensure that new medicines are both safe and effective. Random assignment ensures that treatment and control groups differ only in whether or not they received a candidate drug. Any resulting effects can then be confidently ascribed to the drug, rather than to other differences between groups. In addition, the amount of a drug to which participants are exposed is known with great accuracy. Chemical toxicity studies with laboratory animals also use random assignment and controlled chemical doses.

Due to both practical and ethical concerns, studies of particulate matter and human health do not have the luxury of random assignment or accurate exposure measurement. Instead PM epidemiology is based mainly on "observational studies"—studies in which researchers assess pollution exposure and health outcomes on people as they find them in the real world. This chapter reviews the challenges this creates for the design and interpretation of air pollution health studies.

Key Policy-Related Questions in PM Epidemiology Studies

The ultimate goal of epidemiologic studies is to establish whether there is a genuine causal relationship between a given pollutant and reduced health, and, if so, the scope of the effects and the conditions under which the effects occur. The rest of this section summarizes the specific issues that need to be addressed to make such a determination.

Accounting for non-pollution factors that affect health. Health is affected by a wide range of other factors besides pollution levels, including smoking, income, education, diet, level of physical activity, temperature, humidity and other meteorological factors, etc. These factors are also often correlated with pollution levels. When this happens, the effect of pollution is said to be "confounded," that is, mixed together with the effects of other factors. These other factors are then called "confounders" or "covariates." A study

[40] EPA, "Addendum to the Regulatory Impact Analysis for the NOx SIP Call, FIP, and Section 126 Petitions" (Washington, DC: 1998), EPA, "EPA's Acid Rain Program: Results of Phase I, Outlook for Phase II" (Washington, DC: 2001).

that inadequately accounts for confounding could mistakenly attribute to PM a health outcome that was really caused by some other factor unrelated to air pollution.

To avoid confounding, researchers measure not only pollution levels, but also many potential confounding factors, and use statistical models to remove their effects—a process called "controlling" or "adjusting" for the confounder in question. Any residual relationship that remains between health and air pollution can then more confidently be attributed to a genuine causal relationship, rather than a chance correlation. Nevertheless, it is often impossible to adequately measure and account for all potential confounders, and there is always the risk that a study's results will suffer from "residual confounding"—that is, incomplete accounting for the effects of all important factors that could affect health and that are correlated with air pollution exposure.

Confounding is particularly problematic in air pollution studies. As the effect of interest gets smaller, the potential for confounding becomes greater. The reason is that confounding occurs when a third factor—the confounder—is correlated with both air pollution and health. The chances of this joint correlation having a significant impact on a study increase as the strength of the correlation between air pollution and health decreases. Epidemiologists usually consider a strong effect to be on the order of a factor of two or three increase in the risk of experiencing the health effect of interest.[41] But the putative effects of air pollution are on the order of a few percentage points or less over the typical range of pollutant levels, while the health effects of potential confounders like diet and physical activity are much larger. For example, a major study of the long-term effects of $PM_{2.5}$ exposure reported that a 10 $\mu g/m^3$ increase in long-term $PM_{2.5}$ level increases the risk of an early death by four percent. But for a six foot, 200-pound, non-smoking man, gaining just 15 pounds increases risk of an early death by 17 percent.[42]

What are the responsible pollutants? People are exposed to a wide range of pollutants that could affect health. Exposure varies from person to person based on where people live, how active they are, how much time they spend outdoors, etc. Individual pollutant exposures are almost never directly observed, but are estimated based on centrally located monitoring stations in a given region. Although there are dozens or even hundreds of individual pollutants in the air, data are often available for only six—CO, NOx, SO_2, ozone, $PM_{2.5}$ and PM_{10}. Furthermore, levels of these pollutants are often correlated, sometimes making it difficult to sort out which one is most strongly associated with particular health outcomes. Thus, even if a health effect is caused by air pollution, it can be difficult to determine which pollutant is the culprit. It's therefore important to account for levels of as many pollutants as possible in an epidemiologic analysis, in order to be more certain of which are most associated with particular health effects.

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[41] See, for example, E. L. Wynder, "Epidemiological Issues in Weak Associations," *International Journal of Epidemiology*, vol. 19, suppl. 1 (1990), pp. S5-7, G. Taubes, "Epidemiology Faces Its Limits," *Science*, vol. 269, no. 5221 (1995), pp. 164-9, and E. L. Wynder, "Invited Commentary: Response to Science Article, 'Epidemiology Faces Its Limits'," *American Journal of Epidemiology*, vol. 143, no. 8 (1996), pp. 747-9.

[42] Pope et al., "Lung Cancer, Cardiopulmonary Mortality, and Long-Term Exposure to Fine Particulate Air Pollution," E. E. Calle et al., "Body-Mass Index and Mortality in a Prospective Cohort of U.S. Adults," *New England Journal of Medicine*, vol. 341 (1999), pp. 1097-105.

PM is also made up of several different components whose proportions vary by location and season. PM might affect health regardless of its composition, or there might be particular components of PM—for example, PM emissions from diesel vehicles, sulfate generated from power-plant SO_2 emissions, or metals emitted from industrial mills—that are actually responsible for harm. Understanding which pollutant or mixture of pollutants causes the observed health effects is key for designing pollution control strategies that will actually result in public health improvement.

Are pollutant health effects caused by long-term exposure, short-term exposure, or both? Pollution levels vary from day to day and also over longer periods of time. Pollution can have "acute" effects—harm due to a rise in pollution on a given day that can cause respiratory aggravation or even death in susceptible individuals. However, some diseases, like heart disease and cancer, have very long "latencies"—that is, they develop over a long period of time, on the order of 15 to 20 years. Long-term exposure to high average pollution levels might contribute to the risk of developing such diseases. On the other hand, what appear to be long-term effects might actually be due to an accumulation of acute effects. The implications for policy depend on how pollution affects health.

Is there a threshold, below which pollution causes no harm? Pollution might cause some harm at any exposure, or might not have an effect on health if exposure drops below a particular level, called a "threshold." If a threshold exists, then reducing pollution below the threshold ensures protection of public health from pollution. However, if at least some health damage can occur at any level of a pollutant, then there might be no way to provide complete protection. A concept related to the threshold is the "concentration-response function" (CRF)—the rate at which health effects increase with increases in pollution exposure. A goal of the Clean Air Act is to ensure that air pollution health standards are sufficiently stringent to protect even the most susceptible individuals. If PM has no threshold, then the harm from a given level of PM would be larger than if there were a threshold.

Does pollution shorten life by days, months, or years? If a pollutant shortens life by a matter of days in already-frail individuals who would have died soon in any case—a phenomenon known as "harvesting"—then reducing the pollutant would provide few health benefits. However, if a pollutant can shorten life by months or years in healthy people, then the benefits of pollution reduction would be substantial.

Are proposed health effects biologically plausible? Epidemiologic studies can only identify statistical associations between pollutants and health effects, but cannot by themselves demonstrate a causal connection. Toxicologic studies, in which animals or human volunteers undergo controlled exposures to a pollutant, can help determine whether pollution at levels found in ambient air can actually cause various types of toxic effects, such as inflammation or respiratory distress, and by what biologic mechanisms these effects can occur. Studies of workers occupationally exposed to pollution can also help pin down toxic effects of a given pollutant.

Once the nature and magnitude of health effects is established, the results can feed into an analysis of costs and benefits of various pollution control options.

Health Effects of Long-Term PM Exposure

Health effects from long-term exposure to pollution are usually assessed via types of epidemiologic studies known as "cohort studies" and "ecological studies." Cohort studies follow a cohort of individuals over time. Ecological studies assess the relationship between pollution and health at the group level.

Cohort studies have the advantage of having information on the health status and health-related behaviors of each individual in the study, which allows for more robust control for confounding. Ecological studies have only average information for groups in the study, but not information on each individual. However, in terms of air pollution epidemiology, even cohort studies are partially "ecological" in the sense that much of the data, including air pollution exposure, is available only at the group level, making even nominal cohort studies "semi-ecological."

There are five major U.S. studies of the association between mortality and long-term exposure to PM. Four are semi-ecological cohort studies and one is a fully ecological study.

American Cancer Society (ACS) study.[43] The original ACS cohort study (hereafter referred to as ACSI) included 50 cities and more than 500,000 people, mostly of middle-class socio-economic status. ACSI followed these individuals from 1982 to 1989 and looked at the relationship between measured $PM_{2.5}$ levels and mortality across the cities in the study. ACSI was also the subject of a detailed reanalysis by the Health Effects Institute (HEI), an independent, non-profit research foundation funded by EPA and industry.[44] More recently, the original authors of ACSI, along with some participants in the HEI reanalysis, published another report on the ACS cohort (hereafter referred to as ACSII), this time with a longer follow-up period from 1982 to 1998.[45]

ACSII reported that a 10 $\mu g/m^3$ increase in long-term-average $PM_{2.5}$ levels was associated with a 4 percent increase in the risk of death from 1982 to 1998.[46] The study was based on average $PM_{2.5}$ levels measured in the various cities from 1979 to 1983, which ranged from about 10 to 30 $\mu g/m^3$.[47] By 2000, the range across these cities was about 5 to 20 $\mu g/m^3$. ACSII also reported that chronic PM_{10} exposure was not associated with increased mortality.

A number of features of the various ACS analyses suggest that the reported association of $PM_{2.5}$ with mortality might not represent a genuine cause-effect

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[43] Pope et al., "Lung Cancer, Cardiopulmonary Mortality, and Long-Term Exposure to Fine Particulate Air Pollution," C. A. Pope et al., "Particulate Air Pollution as a Predictor of Mortality in a Prospective Study of U.S. Adults," *American Journal of Respiratory and Critical Care Medicine*, vol. 151, no. 3 Pt 1 (1995), pp. 669-74.

[44] D. Krewski et al., "Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality" (Cambridge, Massachusetts: Health Effects Institute, 2000).

[45] Pope et al., "Lung Cancer, Cardiopulmonary Mortality, and Long-Term Exposure to Fine Particulate Air Pollution."

[46] Ibid.

[47] Ibid.

relationship. For example, ACSI and ACSII assessed health effects using a statistical model that included $PM_{2.5}$ as the only pollutant. But the HEI reanalysis included SO_2 levels in the analysis as a potential confounder and found that the $PM_{2.5}$ effect disappeared. Only SO_2 appeared to be associated with mortality. This strongly suggests that the ACS results suffered from confounding by other pollutants.[48]

Other ACS study results suggest that the apparent association of $PM_{2.5}$ with mortality might instead be a spurious association caused by residual confounding. For example:

- There was no association between $PM_{2.5}$ and mortality for persons with more than a high-school education, for women, and for people between the ages of 60 and 69.[49]
- $PM_{2.5}$ was associated with increased mortality for former smokers, but not current- or never-smokers.
- $PM_{2.5}$ was associated with increased mortality for people who said they were moderately active, but not for people who said they were either sedentary or very active.
- $PM_{2.5}$ was *not* associated with an increase in lung cancer mortality in the HEI reanalysis, which covered the period 1982-1989, but was associated with an increase in mortality due to other cancers.[50]
- When population change was added into the statistical model as a potential confounder, the $PM_{2.5}$ effect declined by two thirds and became statistically insignificant.[51] The hypothesis is that people who leave a city are more likely to

[48] Epidemiologists do not believe that SO_2 at current low levels could be causing harm, but rather that SO_2 may be acting as a surrogate for the pollutant mixture in a given area (see, for example, G. Hoek et al., "Daily Mortality and Air Pollution in the Netherlands," *Journal of the Air and Waste Management Association*, vol. 50, no. 8 (2000), pp. 1380-9, S. H. Moolgavkar, "Air Pollution and Daily Mortality in Three U.S. Counties," *Environmental Health Perspectives*, vol. 108, no. 8 (2000), pp. 777-84, and F. W. Lipfert, "Commentary on the HEI Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality," *Journal Toxicology and Environmental Health, Part B,* in press). Current SO_2 levels are 50 percent below those of 1980 and 75 percent below those of the 1960s. 98 percent of monitoring locations never reach SO_2 levels of even half the federal health standard (current SO_2 levels are based on author's analysis of national SO_2 monitoring data downloaded from EPA's AIRData Web site, www.epa.gov/aqspubl1/select.html. SO_2 trends since 1980 come from EPA, "Latest Findings on National Air Quality: 2000 Status and Trends." Pre-1980 trends come from Goklany, *Clearing the Air*, Figure 3-2. The pre-1980 data are based on only 21 monitoring locations, while more recent data are based on several hundred locations).

[49] When cardiopulmonary and lung cancer mortality were looked at separately, both men and women had an increased risk of the former, while only men had an increased risk of the latter.

[50] See Table 20 in Krewski et al., "Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality." ACSII did find an association between $PM_{2.5}$ and lung-cancer mortality for the period 1982-1998. However, even this association held only for men, those with no more than a high-school education, and those not in the 60-69 age range.

[51] See Table 37 in Ibid. The term "statistically significant" is a term of art in statistical analysis used to signify a result that is considered, based on objective criteria, unlikely to have occurred by chance due to random variability in the data. The word "significant" in this context does not in any way mean "important" or "noteworthy" as it would in everyday use. In addition, simply because a result is statistically significant does not mean that it represents a "real" effect, because the underlying data or statistical model could suffer

be healthier than people who remain behind. Cities that lost population—Midwest "rust belt" cities—also had higher $PM_{2.5}$ levels on average. Thus, the apparent effect of $PM_{2.5}$ could actually have resulted from a reduction in the average health of residents caused by healthier people moving away from areas of the country that were in economic decline.

These odd variations in the relationship between $PM_{2.5}$ and mortality appear to be biologically implausible and suggest that other factors besides pollution would better explain the results. In addition, the ACS study reported that higher $PM_{2.5}$ levels were *not* associated with an increased risk of mortality due to respiratory disease; a surprising finding, given that PM would be expected to exert its effects through the respiratory system.[52]

Another concern with the ACS study is that information about participants' health-related behaviors and status, such as diet, body-mass index (BMI; a measure of relative body size) and smoking were assessed only in 1982 when they entered the study, but not afterward. If any of these factors changed after 1982, and if the changes were correlated with pollution levels, then the study results would suffer from additional uncontrolled confounding. For example, if people living in areas with higher pollution were also either more likely to get fatter, or less likely to stop smoking between 1982 and 1998 when compared with people in lower-pollution areas, researchers could mistake an effect of body weight or smoking for an effect of air pollution. The rate of BMI increases or smoking decreases and the likelihood of living in an area of greater air pollution are probably positively correlated through their common association with socio-economic factors such as income and education, suggesting this is a concern worth additional investigation.[53]

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from various kinds of bias (e.g., confounding), which are a much larger source of uncertainty in epidemiologic studies than the effect of random variation in the data. Statistical significance is thus generally considered a necessary, but not sufficient condition for a statistical result to be considered as genuinely representing some underlying real feature of the world.

[52] See Table 20 in Ibid.

[53] According to the Centers for Disease Control, Americans' average BMI has indeed increased substantially during the last 20 years, and poorer people and minorities are at greater risk for obesity than whites and wealthier people. People with less education were less likely to stop smoking during the last 20 years when compared with more educated people. Minorities are more likely to live in areas with more particulate pollution. Thus, there is a significant potential for changes in BMI, smoking or other health-related behaviors to be mistaken for an effect of air pollution through their common association with socio-economic factors. (Sources: Obesity: National Center for Health Statistics, "Health, United States, 1998, with Socio-Economic Status and Health Chartbook," Centers for Disease Control, 1999, www.cdc.gov/nchs/data/hus/hus98.pdf; Smoking: National Center for Health Statistics, "Health, United States, 2001," Centers for Disease Control, 2001, www.cdc.gov/nchs/data/hus/hus01.pdf; Air Pollution: National Center for Health Statistics, "Health, United States, 1998," and Victor Brajer and Jane V. Hall, "Recent Evidence on the Distribution of Air Pollution Health Effects," *Contemporary Policy Issues*, vol. 10 (April 1992), pp. 63-71).

Because the risks of smoking and obesity are so much larger than the risk the ACS study estimated for $PM_{2.5}$, even a small difference in smoking and obesity trends between areas with differing pollution levels could swamp the ostensible effect of differences in air pollution. For example, ACSII found that a 10 $\mu g/m^3$ increase in $PM_{2.5}$ increases mortality risk by 4 percent. But for a six foot, 200-pound, non-smoking man,

Long-term studies are based on the hypothesis that chronic exposure to elevated pollution causes the development of cardiovascular disease or cancer. These diseases have latencies of 15 to 20 years between exposure and manifestation of disease, suggesting that pollution exposure should be measured during a time period years before the health effect appears. Yet the ACS pollution measurements occurred around the same time the study began in early 1980s, and the range of PM levels was about four times higher during the 1960s than during the 1980s.[54] If it was these earlier high PM levels that actually caused the health effects, then the real effect of air pollution would be one-fourth that estimated in the ACS study. This is because studies like ACS estimate the concentration-response function for PM health effects based on the range of PM levels across cities in the study. If this range is actually four times greater than the range used in the ACS study, then the health effects of a given increase in PM would be one-fourth of what the ACS study estimated.[55]

The ACS results also suggest that $PM_{2.5}$ risks are decreasing with time. ACSI reported that a 10 $\mu g/m^3$ increase in $PM_{2.5}$ was associated with a 6.9 percent increase in mortality for the period 1982-1989. But this risk declined to 2.5 percent for 1990-1998 period, or 64 percent lower than for 1982-1989.[56] The PM-mortality relationship for 1990-98 is also statistically insignificant.[57]

Harvard Six Cities (HSC) study.[58] This cohort study compared chronic mortality data with annual-average $PM_{2.5}$ levels in six cities located in the Midwest and northeast. $PM_{2.5}$ measurements were collected from the late 1970s through the mid-1980s, and mortality data were based on a 14- to 16-year follow-up of about 8,000 individuals. The HSC study was also the subject of a detailed reanalysis by HEI.[59]

HSC found, after adjusting for confounders such as smoking and educational attainment, that there was a 26 percent increase in risk of death between the city with the highest mean $PM_{2.5}$ level (29.6 $\mu g/m^3$) and the lowest (11 $\mu g/m^3$). This works out to a mortality increase of 14 percent for each 10 $\mu g/m^3$ increase in $PM_{2.5}$—substantially larger than that found in any of the other long-term mortality studies.

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gaining just 15 pounds increases his risk of an early death by 17 percent (Calle et al., "Body-Mass Index and Mortality in a Prospective Cohort of U.S. Adults").

[54] Lipfert, "Commentary on the HEI Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality."

[55] Ibid.

[56] Pope et al. did not point out this key feature of their analysis in their published results. They reported only results for 1982-1989 (in ACSI) and 1982-1998 (in ACSII). However, the results for 1990-1998 can be inferred from the data presented ACSI and ACSII.

[57] The fact that the 1990-1998 PM-mortality relationship is statistically insignificant can be inferred from the magnitude of the PM-mortality relationship for 1990-1998 and the 95 percent confidence intervals reported for the other time periods.

[58] D. W. Dockery et al., "An Association between Air Pollution and Mortality in Six U.S. Cities," *New England Journal of Medicine*, vol. 329, no. 24 (1993), pp. 1753-9.

[59] Krewski et al., "Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality."

Although the study found a mortality increase between the highest and lowest $PM_{2.5}$ cities (Steubenville, OH and Portage, WI, respectively), the increase in mortality for the other four cities when compared with Portage was not statistically significant. This is noteworthy, because after Steubenville, the next highest $PM_{2.5}$ level was 20.9 $\mu g/m^3$ for Harriman, TN. Based on national $PM_{2.5}$ data for 1999-2001, less than 2 percent of monitoring locations have annual mean $PM_{2.5}$ levels greater than 21 $\mu g/m^3$.[60] Taking the results of the HSC study at face value, this suggests that very few areas of the country now have $PM_{2.5}$ levels associated with increases in mortality due to long-term exposures.

There is also evidence that the HSC results suffer from residual confounding. For example, HSC did not account for physical activity level of the study participants, yet exercise is strongly correlated with health. It turns out that levels of physical activity in the six cities are inversely correlated with pollution levels in these cities.[61] HSC might therefore have attributed to air pollution a health effect that was actually caused by lower physical activity levels. Like the ACS study, there was no association between $PM_{2.5}$ and mortality in people with more than a high-school education. HSC also found that greater $PM_{2.5}$ was associated with a statistically insignificant *decrease* in mortality due to respiratory causes specifically.

The HSC study was based on $PM_{2.5}$ levels measured concurrent with the beginning of the follow-up period, even though mortality was due to diseases with long latency times. Therefore, like the ACS study, the HSC study might therefore have inflated the apparent effect of $PM_{2.5}$ on mortality, compared to an assessment based on much greater $PM_{2.5}$ levels in the two decades leading up to the HSC follow-up period.

Because HSC included only six locations, it was not possible to investigate whether including other pollutants in the statistical analysis affected the apparent mortality contribution of $PM_{2.5}$.

Washington University-EPRI Veterans study (Veterans study).[62] The Veterans' study assessed the relationship between $PM_{2.5}$ and mortality in 50,000 male U.S. veterans. The study population included men with preexisting high blood pressure, which should have made them more susceptible to the effects of PM, and a 21-year follow-up period. Data on total suspended particulates (TSP) were available dating back to 1953, while $PM_{2.5}$ data were available for the period 1979-84. Unlike the ACS and HSC studies, the Veterans study assessed associations between PM and mortality for several time periods, and assessed both concurrent and delayed health effects of pollution exposure.

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[60] Of the 19 monitoring locations in the U.S. that fall into this category, 17 are in southern California and California's Central Valley. None of the cities in the HSC study currently have $PM_{2.5}$ levels above 21 $\mu g/m^3$. Steubenville is still the highest at 19.3 $\mu g/m^3$.

[61] F. W. Lipfert, "Estimating Air Pollution-Mortality Risks from Cross-Sectional Studies: Prospective vs. Ecologic Study Designs," Health and Regulatory Issues, Proceedings of the International Specialty Conference, Air and Waste Management Association, 1995.

[62] F. W. Lipfert et al., "The Washington University-EPRI Veterans' Cohort Mortality Study," *Inhalation Toxicology*, vol. 12 (suppl. 4) (2000), pp. 41-73.

The study found a statistically significant *decrease* in mortality associated with $PM_{2.5}$. When various ecological confounding variables were added to the statistical analysis, $PM_{2.5}$ was associated with an even greater reduction in mortality. While it is not plausible that higher $PM_{2.5}$ could improve health, this study suggests that chronic exposure to elevated $PM_{2.5}$ is not associated with increases in mortality. The reported associations between mortality and pollution were greatest for pollution exposures occurring within a few years of death, which is unexpected if $PM_{2.5}$ is causing diseases with long latency periods, such as cancer and heart disease.

The Veterans study did not control for diet and exercise. In addition, the study also assessed people only at entry, so some personal characteristics may have changed. As a result there may be some residual confounding that could explain the anti-correlation between $PM_{2.5}$ and health. Nevertheless, this study's statistical analysis of individual health factors is more comprehensive than that of the ACS or HSC, because it includes other non-pollution health-related factors, such as age, smoking-status, blood pressure, and body-mass index. Further, these factors had the expected association with mortality (e.g., high blood pressure was associated with increased risk of death), making it more difficult to discard the pollution results. The study assessed the effect of $PM_{2.5}$ alone, and was not able to determine whether adding other pollutants to the analysis would change the apparent $PM_{2.5}$ effect.

Because this study assessed only male veterans with high blood pressure, the results might not hold for the U.S. population in general. However, one would expect that the study group would be *more* susceptible to PM-induced health effects than the general population.

Adventist Health Study of Smog (AHSMOG).[63] AHSMOG followed a cohort of about 6,300 white, non-smoking Seventh Day Adventists in California from 1977 to 1992, and assessed the association of PM_{10} with mortality. The study found that a 20 $\mu g/m^3$ increase in the average PM_{10} level was associated with a 9 percent increase in mortality in males, but the increase was not statistically significant. PM_{10} had no association with mortality in females.

AHSMOG also assessed whether frequent exposure to high daily PM levels was associated with mortality. In this case the study found a statistically significant 12 percent increase in male mortality when PM_{10} exceeded 100 $\mu g/m^3$ on at least 43 days per year. Once again, there was no effect in females.

These results are based on past PM_{10} levels, which were much greater than current levels. For example, only about one percent of U.S. PM_{10} monitoring locations, most in southern California and California's Central Valley, now exceed 100 $\mu g/m^3$ on more than 37 days per year.[64]

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[63] D. E. Abbey et al., "Long-Term Inhalable Particles and Other Air Pollutants Related to Mortality in Nonsmokers," *American Journal of Respiratory and Critical Care Medicine*, vol. 159, no. 2 (1999), pp. 373-82.

[64] Author's analysis of national PM_{10} monitoring data downloaded from EPA's AIRData Web site, www.epa.gov/aqspubl1/select.html. I was not able to assess the 43rd highest reading directly, as the closest

County-based ecological study (County study).[65] This fully ecological study included all U.S. counties with air pollution monitoring data, and assessed the relationship between pollution levels and mortality at the county level between 1960 and 1997. Like the Veterans study, this study also assessed the relationship between pollution and mortality for several time periods, and assessed both concurrent and delayed health effects of pollution exposure.

The County study found an association between greater $PM_{2.5}$ and increased mortality. However, there appeared to be a threshold somewhere between 20 and 25 $\mu g/m^3$, below which $PM_{2.5}$ had no effect. In addition, the relationship between pollution and mortality was strongest when pollution exposure occurred within a few years of death. There was little or no evidence for cumulative effects from longer-term pollution exposure.

In a comparison with counties that were part of the HSC, this study found that $PM_{2.5}$ was associated with an increase in mortality only for Steubenville, and that the threshold $PM_{2.5}$ level for mortality increases was at least 23 $\mu g/m^3$.

When looking at different age groups, the health effects of pollution were larger for younger age groups. This argues against chronic effects, because effects should be greater for people with more cumulative exposure. Like the ACS study, the County study did not find a mortality risk associated with PM_{10}.

This study included a wider range of non-pollutant confounders in the analysis when compared with other studies of long-term mortality, and found the expected directions for their effects, also adding weight to the validity of the estimated pollution effects.

Responsible Components of PM

Some of these studies also assessed the effects of long-term exposure specifically to the sulfate component of PM. Sulfate is created mainly from gaseous SO_2 emissions from power plants and other industrial sources in the eastern half of the United States. However, the epidemiologic results for sulfate suffer from the same concerns as for PM as a whole.

For example, in the ACS study, sulfate appeared to have a substantial protective effect against death due to respiratory causes that almost reached statistical significance. The relationship of sulfate particles to mortality became statistically insignificant when either SO_2 or population change were included in the statistical model, and the sulfate effect dropped to zero when multiple confounders were added to the analysis.[66] The Veterans study found an inverse relationship between sulfate and mortality, while the County study found small risks from sulfate in the 1960s and 1970s that declined to zero

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value easily available from EPA is the 10^{th} percentile of daily PM_{10} readings for each year. This is equivalent to roughly the 37^{th} highest daily PM_{10} reading in a given year.

[65] F. W. Lipfert and Morris, "Temporal and Spatial Relations between Age Specific Mortality and Ambient Air Quality in the United States: Regression Results for Counties, 1960-97."

[66] Krewski et al., "Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality," see Table 20, page 158, and Table 34, page 180.

during the 1990s.[67] The AHSMOG study did not find a statistically significant increase in risk due to sulfates. Therefore, to the extent PM or one of its components is associated with mortality due to long-term exposure, sulfate doesn't seem to be a good candidate for the causal factor.[68]

Summary of Long-Term PM Effects

The evidence suggests that long-term PM exposure at current levels is unlikely to increase risk of death. The ACS and HSC studies suffer from confounding from other pollutants and non-pollution factors that call into question their claimed association between long-term $PM_{2.5}$ exposure and mortality. The Veterans and County studies suggest that $PM_{2.5}$ either has no effect at current or past levels, or that the threshold for harm is somewhere above 20 $\mu g/m^3$—a level exceeded in only a few locations, mainly in parts of California.

Health Effects of Short-Term PM Exposure

The previous section assessed whether long-term exposure to PM might increase the prevalence of deadly diseases that develop over time. In this section, we look at the potential for daily variation in PM levels to cause acute increases in mortality. There is no way to pin down a one-to-one relationship for any given person between death and daily air pollution levels. Therefore, researchers use epidemiologic methods to look for statistical associations between daily variation in pollution levels and the rate of various health outcomes among residents within a community or region. These studies are always ecological, because both air pollution exposure and health effects are assessed at the group, rather than individual, level.

Researchers have performed dozens of studies to assess whether acute changes in daily air pollution levels can cause death or disease.[69] Based on the results of these studies, the conventional wisdom has been that typical daily changes in $PM_{2.5}$ and PM_{10}—on the order of up to tens of micrograms per cubic meter—change rates of death and hospitalization by up to a few percentage points. This might seem like a small effect, and indeed an effect of this size suggests that air pollution accounts for a tiny fraction of all death and disease. However, when multiplied by tens of millions of people in a population, this result suggests PM could be killing tens of thousands of people per year and causing respiratory distress to hundreds of thousands. A number of recent developments have, however, raised serious concerns over the validity of these results, which I review below.

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[67] Lipfert and Morris, "Temporal and Spatial Relations between Age Specific Mortality and Ambient Air Quality in the United States: Regression Results for Counties, 1960-97," and Lipfert et al., "The Washington University-EPRI Veterans' Cohort Mortality Study."

[68] As will be discussed below, toxicologic results also suggest that sulfate is a poor candidate for the harmful component of PM.

[69] See Chapter 8 of EPA (2002) and Lipfert (2002) for a detailed listing of relevant studies (EPA, "Air Quality Criteria for Particulate Matter, Third External Review Draft" (Washington, DC: 2002), and F. W. Lipfert, "Review Comments on the U.S. Environmental Protection Agency's Air Quality Criteria for Particulate Matter, Third External Review Draft" (Annapolis, MD: Annapolis Center for Science-Based Public Policy, 2002).

It is also worth remembering that hardly any monitoring locations exceed EPA's daily standards for PM_{10} or $PM_{2.5}$, so for current policy purposes the question of deaths due to daily PM increases is somewhat moot. However, there is still the substantive and important issue of whether PM at levels below the EPA standards could be causing harm, which would bolster the case for more stringent daily PM standards.

Software Glitches

The National Mortality and Morbidity Air Pollution Study (NMMAPS) is arguably the most comprehensive analysis of the acute effects of PM_{10} on health.[70] Funded and overseen by the Health Effects Institute and performed by researchers from Johns Hopkins and Harvard, NMMAPS assessed the relationship between PM_{10} and daily mortality in 90 U.S. cities, and PM_{10} and hospital admissions in 14 cities.

By pooling the results from the 90 cities in the study, NMMAPS estimated that a 10 $\mu g/m^3$ increase in daily PM_{10} levels increases daily deaths by 0.41 percent. However, early in 2002 the NMMAPS researchers discovered a software glitch that caused this result to be spuriously high.[71] After correcting the error, the new estimate is 0.27 percent—34 percent lower than the original estimate. Using a different statistical technique, the estimate declined further, to 0.21 percent.[72]

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[70] J. M. Samet et al., "The National Morbidity, Mortality, and Air Pollution Study. Part II: Morbidity and Mortality from Air Pollution in the United States," *Research Report / Health Effects Institute*, no. 94, pt. 1 (2000), pp. 5-70; discussion 71-9, and J. M. Samet et al., "The National Morbidity, Mortality, and Air Pollution Study. Part I: Methods and Methodologic Issues," *Research Report / Health Effects Institute*, no. 94, pt. 2 (2000).

[71] The details of the problem are quite technical, but the basic idea is as follows: Statistical software packages come with default settings for the level of precision required in any given set of statistical calculations. These default settings are appropriate for the vast majority of users. However, in air pollution epidemiology researchers are assessing exceedingly small effects—on the order of a one percent change or less. The default precision settings in statistical software are typically set at about this same level of precision. However, to ensure valid results the default settings need to be at no more than a small fraction of the size of the effect being measured. As a result of this problem, acute-effects air pollution studies published during the last several years might have in effect failed to control for confounding.

The particular problem identified here is a special case of what might be a more general problem in the PM epidemiology literature. Recent studies on the acute effects of PM and other air pollutants use relatively new, computationally intensive statistical techniques. Such techniques are prone to numerical inaccuracy when implemented on a computer, because computers must use "floating-point arithmetic" for computations. This means that computers can carry only a certain number of decimal places in the numbers used for successive computations. Computations that involve many iterations, as the epidemiological techniques do, can turn small numerical inaccuracies into large ones. These effects are unimportant in most applications, but can become dominant when the real effect is small, as it is in the epidemiologic studies. Econometricians have been documenting numerical inaccuracies of various statistical software packages, but it appears that no one has yet checked the extent to which they might have affected the results of published epidemiologic studies (A. E. Smith and T. H. Savage, "Comments on the Environmental Protection Agency's Third External Review Draft of Air Quality Criteria for Particulate Matter" (Washington, DC: Charles River Associates, 2002)).

[72] The NMMAPS authors have posted their updated results at www.biostat.jhsph.edu/biostat/research/nmmaps_faq.htm.

This software problem potentially affects dozens of air pollution health studies that used the same methods and the same or similar software. As a result, EPA, other agencies, and epidemiologists are reevaluating the acute-effects air pollution epidemiology literature.[73] The software issue has exacerbated concerns about the specific methods and results used to support calls for tougher daily PM standards. The sections below review these concerns.

Confounding

As with the long-term studies, studies of the relationship between daily changes in PM levels and mortality can suffer from confounding due to inadequate control for either other pollutants or non-pollution factors that are correlated with both health and air pollution. Many studies of the acute effects of PM on health have considered only PM, but not levels of other pollutants. Studies that employed "multi-pollutant" models have often found that the apparent effect of PM is greatly diminished or disappears completely when other pollutants are considered.

For example, a number of studies have variously found that SO_2, CO, or NO_2 diminish the apparent PM effect when added to models of acute air pollution effects. A study of daily mortality in Los Angeles, Chicago, and Phoenix from 1987 to 1995 found that CO was much more strongly associated with mortality than were particles. $PM_{2.5}$ had no association with mortality in Los Angeles when CO was included in the analysis, while the effect of PM_{10} was diminished or removed entirely when various gaseous pollutants were included.[74] Some multi-city studies in Canada and Europe have found similar results.[75] The new NMMAPS result reported above was not adjusted for the effects of other pollutants, and may therefore overestimate the apparent effect of PM_{10} on health. On the other hand, there are also multi-pollutant studies that have found that the PM effect remains even after including gaseous pollutants in the statistical model.[76]

A recent meta-analysis[77] of studies of pollution and acute mortality found that including one or more additional pollutants in a statistical analysis generally diminished the apparent effect of the first pollutant alone, often rendering it statistically insignificant. However, when all the studies were pooled, PM_{10} and SO_2 were still associated with a

[73] For a list of studies suggested for review, see L. Grant, letter to Philip Hopke, Chair, Clean Air Scientific Advisory Committee, and Smith and Savage, "Comments on the Environmental Protection Agency's Third External Review Draft of Air Quality Criteria for Particulate Matter."

[74] Moolgavkar, "Air Pollution and Daily Mortality in Three U.S. Counties." $PM_{2.5}$ data were available only for Los Angeles, so this analysis was not performed for the other two cities.

[75] See, for example, R. T. Burnett et al., "The Effect of the Urban Ambient Air Pollution Mix on Daily Mortality Rates in 11 Canadian Cities," *Canadian Journal of Public Health*, vol. 89, no. 3 (1998), pp. 152-6, and Hoek et al., "Daily Mortality and Air Pollution in the Netherlands."

[76] See, for example, K. Katsouyanni et al., "Short-Term Effects of Ambient Sulphur Dioxide and Particulate Matter on Mortality in 12 European Cities: Results from Time Series Data from the APHEA Project. Air Pollution and Health: A European Approach," *British Medical Journal*, vol. 314, no. 7095 (1997), pp. 1658-63.

[77] Meta-analysis is a statistical technique in which results from many different studies are combined in an effort to identify consistent overall results.

statistically significant increase in mortality.[78] Even so, when it comes to mixtures of air pollutants, it is not possible to control for confounding in the traditional sense. There are dozens of pollutants in ambient air, measurements are available for only a few, and most "multi-pollutant" studies have included no more than two or three pollutants in their analyses. Furthermore, pollutants that appear to have the greatest association with health effects are often present at such low levels that they probably could not actually be causing harm.[79] This has led to the suggestion that whatever pollutant appears most associated with health effects might be acting as a surrogate marker for the effects of the particular pollution mix in a given area, and that epidemiologic studies are not capable of determining which specific pollutant(s) is(are) causing observed health effects.[80] This is consistent with the observation that the magnitude of the association of pollution with mortality is similar across all pollutants studied.

Non-pollution factors create a potentially even more serious confounding problem. While the putative health effects of the various pollutants are of similar magnitude at current ambient levels, the health effects of some confounders, including weather and season, can be much larger than the pollution effects.[81] Improperly accounting for these non-pollution effects could cause one to mis-attribute health effects to pollution that were in fact caused by weather.[82]

Most studies of pollution and daily mortality published before the mid-1990s may have failed to adequately account for key confounders, making their results potentially invalid.[83] More recent studies have found that accounting for all the important confounding factors can be difficult and often leads to a reduction in the apparent health effects of PM.[84] For example, NMMAPS reported that higher ozone was associated with

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[78] D. M. Stieb et al., "Meta-Analysis of Time-Series Studies of Air Pollution and Mortality: Effects of Gases and Particles and the Influence of Cause of Death, Age, and Season," *Journal of the Air and Waste Management Association*, vol. 52, no. 4 (2002), pp. 470-84.

[79] See, for example, Moolgavkar, "Air Pollution and Daily Mortality in Three U.S. Counties," and Hoek et al., "Daily Mortality and Air Pollution in the Netherlands."

[80] Moolgavkar, "Air Pollution and Daily Mortality in Three U.S. Counties," S. H. Moolgavkar, "Review of Chapter 8 of the Criteria Document for Particulate Matter (Comments Submitted to EPA)" 2002, and F. W. Lipfert et al., "Daily Mortality in the Philadelphia Metropolitan Area and Size-Classified Particulate Matter," *Journal of the Air and Waste Management Association*, vol. 50, no. 8 (2000), pp. 1501-13.

[81] P. Switzer, "A Review of Statistical Methods Used in Time-Series Epidemiologic Studies of Ambient Particulate Matter and Acute Health Effects Cited by the April 2002 EPA Draft PM Criteria Document" (Palo Alto, California: Stanford University, 2002).

[82] See, for example, E. Hennessy, "Air Pollution and Short Term Mortality," *British Medical Journal*, vol. 324, no. 7339 (2002), pp. 691-2, and R. L. Smith et al., "Regression Models for Air Pollution and Daily Mortality: Analysis of Data from Birmingham, Alabama," *Environmetrics*, vol. 11 (2000), pp. 719-43.

[83] S. H. Moolgavkar and E. G. Luebeck, "A Critical Review of the Evidence on Particulate Air Pollution and Mortality," *Epidemiology*, vol. 7, no. 4 (1996), pp. 420-8, and Smith and Savage, "Comments on the Environmental Protection Agency's Third External Review Draft of Air Quality Criteria for Particulate Matter."

[84] See, for example, Smith et al., "Regression Models for Air Pollution and Daily Mortality: Analysis of Data from Birmingham, Alabama," Hennessy, "Air Pollution and Short Term Mortality," and P. Switzer, "Estimating Separately Personal Exposure to Ambient and Nonambient Particulate Matter for

increased mortality in summer, but with reduced mortality in winter, something that is not biologically plausible. Mortality rises in all climates in winter and also during summer heat waves. But ozone is also at its highest during summer heat waves and lowest during winter. This indicates that the NMMAPS results might suffer from inadequate accounting for the effects of seasonal changes in mortality unrelated to air pollution.[85]

The very nature of the mathematical techniques used in epidemiology can also make it difficult to sort out which pollutants or non-pollutant confounders are actually responsible for observed health effects. Epidemiologic studies use a statistical technique called regression analysis to identify which factors are most associated with health outcomes.

There are two technical issues here: First, the mathematical properties of regression are such that factors that have greater variation over time or space will appear to be more strongly associated with health outcomes, regardless of the intrinsic hazard caused by the factor in question.[86] In other words, given two pollutants that are equally toxic at ambient levels, a regression analysis will nevertheless spuriously suggest that the more variable pollutant has a greater effect on health. Ozone, $PM_{2.5}$, and acidic aerosols are more variable than PM_{10} or NO_2. The same concern applies to non-pollutant factors that affect health and often vary greatly from day to day, such as temperature and humidity.

Second, there is error associated with measurement of all pollutants and non-pollutant factors. This error comes from both random error in the measurements themselves and also error associated with using a single monitoring location to characterize air pollution exposure for people all over a city who spend varying amounts of time outdoors and have varying levels of physical activity. In a regression analysis, if two pollutants have an equal intrinsic effect on health, the one measured with the least error will spuriously appear to have a larger effect on health.[87] For example, some studies have reported a greater effect of $PM_{2.5}$ on health than that attributed to coarser particles.[88] There is good reason to believe that measurement error is greater for coarse particles than for fine particles, which would tend to make $PM_{2.5}$ spuriously appear more toxic than coarse

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Epidemiology and Risk Assessment: Why and How," *Journal of the Air and Waste Management Association*, vol. 51, no. 3 (2001), pp. 322-3; discussion 29-38.

[85] Lipfert, "Review Comments on the U.S. Environmental Protection Agency's Air Quality Criteria for Particulate Matter, Third External Review Draft."

[86] F. W. Lipfert and R. E. Wyzga, "Air Pollution and Mortality: The Implications of Uncertainties in Regression Modeling and Exposure Measurement," *Journal of the Air and Waste Management Association*, vol. 47, no. 4 (1997), pp. 517-23.

[87] F. W. Lipfert and R. E. Wyzga, "Statistical Considerations in Determining the Health Significance of Constituents of Airborne Particulate Matter," *Journal of the Air and Waste Management Association*, vol. 49, no. 9 (1999), pp. 182-91.

[88] See, for example, J. Schwartz et al., "Is Daily Mortality Associated Specifically with Fine Particles?" *Journal of the Air and Waste Management Association*, vol. 46, no. 10 (1996), pp. 927-39, and R. J. Klemm et al., "Is Daily Mortality Associated Specifically with Fine Particles? Data Reconstruction and Replication of Analyses," *Journal of the Air and Waste Management Association*, vol. 50, no. 7 (2000), pp. 1215-22.

material even if their real effects are the same.[89] This too makes it difficult to determine which pollutant(s) or non-pollutant factor(s) are actually responsible for observed health outcomes.

A recent assessment of the difficulties in sorting out these issues in air pollution epidemiology studies concluded that, "Estimation of very weak associations in the presence of measurement error and strong confounding is inherently challenging. In this situation, prudent epidemiologists should recognize that residual bias can dominate their results."[90]

Heterogeneity of Effects Among Cities

As noted earlier, NMMAPS pooled the results from 90 cities to arrive at a single estimate of the effect of daily PM_{10} levels on mortality. But the pooled estimate glosses over the considerable variation in results from city to city. In 32 of the 90 cities, increases in PM were associated with a *decreased* risk of mortality, and the protective effect was statistically significant for one of the cities (Little Rock).[91] Among the 58 cities where PM was associated with increased mortality, the effect was statistically significant for only two cities (New York and Oakland).[92] A number of other multi-city studies have also found substantial variability of estimated effects in different locations.[93]

This weakens the case for current PM levels as a cause of increased mortality, and also suggests that a pooled average mortality rate from NMMAPS or other studies may have no real meaning. Pooling results across locations is only justified when measuring the same effect in different regions. The large variation from city to city suggests that different factors might be at work in different places, and that PM is acting as a surrogate for different mixes of health-related factors in different cities.[94]

The NMMAPS results also highlight the effect of outliers on the overall estimate of PM health effects. As noted earlier, NMMAPS reported that only New York and Oakland had a statistically significant increase in mortality associated with PM_{10}, while Little Rock had a statistically significant decrease in mortality. When these three outliers are removed from the analysis, the estimated average risk for a 10 $\mu g/m^3$ increase in PM_{10}

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[89] Lipfert and Wyzga, "Air Pollution and Mortality: The Implications of Uncertainties in Regression Modeling and Exposure Measurement."

[90] T. Lumley and L. Sheppard, "Time Series Analyses of Air Pollution and Health: Straining at Gnats and Swallowing Camels?" *Epidemiology*, vol. 14, no. 1 (2003), pp. 13-4.

[91] This doesn't mean that PM_{10} should be considered beneficial to health in these cities, but it does make it unlikely that PM_{10} was detrimental, and also suggests that important health-related factors are missing from the epidemiological modeling.

[92] NMMAPS used exactly the same statistical model for all 90 cities, so the large differences between cities can't be due to differences in modeling strategy.

[93] See, for example, Moolgavkar, "Air Pollution and Daily Mortality in Three U.S. Counties," Katsouyanni et al., "Short-Term Effects of Ambient Sulphur Dioxide and Particulate Matter on Mortality in 12 European Cities: Results from Time Series Data from the APHEA Project. Air Pollution and Health: A European Approach."

[94] Moolgavkar, "Review of Chapter 8 of the Criteria Document for Particulate Matter (Comments Submitted to EPA)."

across the remaining 87 cities declines from 0.22 to 0.15 percent. Since the 0.22 percent result is barely statistically significant, removing the three outlier-cities presumably also causes the newly estimated overall PM_{10} effect to become statistically insignificant.[95]

Variability of Results Among Different "Models"

The process of estimating the health effects of air pollution involves developing a mathematical equation or "model" intended to represent the key real-world features of the relationship between pollution and health. In research parlance, the development of this model is known as "model specification." In addition to uncertainties in the data that go into the model, the structure of the model itself is a source of considerable uncertainty in air pollution studies. Subtle variations in the structure of statistical models of air pollution's health effects can have great influence on the estimated effect of PM on health.

A recent review on model uncertainty in PM studies noted that modeling "is often done in a highly exploratory fashion, and different model selection strategies may lead to different models and conclusions about the magnitude of relative risks associated with changes in particulate matter…For making inferences, the selected 'best' model is often treated as if it were the true model. This procedure ignores the uncertainty involved in model selection, and may lead to overconfident predictions and policy decisions that are riskier than one thinks they are…Model uncertainty often outweighs other sources of uncertainty, but is typically ignored in practice."[96] Specific issues include:[97]

- **Overall modeling approach.** There is a wide array of modeling techniques corresponding to different mathematical forms for the equation relating PM to mortality or other health outcomes. The details of these different approaches are technical and beyond the scope of this paper. However, the degree to which daily PM levels appear related to health depends on the specifics of the chosen model.[98]
- **Definition of PM exposure.** Study results vary based on how PM exposure is defined. For example, mortality might depend on PM levels today, yesterday, the day before yesterday, etc., or on some average of PM levels during the last few days. This is known as the "lag structure" of the model, because mortality is expected to follow or "lag" an increase in PM levels.

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[95] Ibid.

[96] M. Clyde, "Model Uncertainty and Health Effect Studies for Particulate Matter," *Environmetrics*, vol. 11 (2000), pp. 745-63.

[97] On these issues, see, for example, Ibid., Smith et al., "Regression Models for Air Pollution and Daily Mortality: Analysis of Data from Birmingham, Alabama," Moolgavkar and Luebeck, "A Critical Review of the Evidence on Particulate Air Pollution and Mortality," Lipfert and Wyzga, "Air Pollution and Mortality: The Implications of Uncertainties in Regression Modeling and Exposure Measurement," and Switzer, "A Review of Statistical Methods Used in Time-Series Epidemiologic Studies of Ambient Particulate Matter and Acute Health Effects Cited by the April 2002 EPA Draft PM Criteria Document."

[98] See, for example, Smith et al., "Regression Models for Air Pollution and Daily Mortality: Analysis of Data from Birmingham, Alabama," and S. H. Moolgavkar et al., "Particulate Air Pollution, Sulfur Dioxide, and Daily Mortality: A Reanalysis of the Steubenville Data," *Inhalation Toxicology*, vol. 7 (1995), pp. 35-44.

Researchers often test several different lag structures because no one knows what the genuine temporal relationship is between exposure to PM and changes in health. Different lag structures lead to different conclusions regarding whether increases in PM can harm health. Furthermore, results vary from city to city as to which lag structure results in the greatest apparent PM effect. This appears to be inconsistent with the expectation that PM would have similar health effects in different locations, and may suggest inadequate control for confounding.[99]

In studies that consider multiple lags, researchers often select the lag that gives the largest PM effect. This creates an upward bias in estimates of PM health effects, because random variability in the data can result in high PM effects at particular lags that are due to chance alone. For example, a recent simulation study found that, even if PM has no real effect on health, picking only the lag with the maximum PM effect gives a result of about the same magnitude as is typically reported in PM acute effects studies.[100]

- **Choice of monitoring locations used to represent PM exposure.** Results of PM studies vary by which monitoring locations in a given region are chosen to represent PM exposure. For example, choosing different monitors or groups of monitors in a city to represent the PM exposure of city residents results in different estimates of PM health effects.[101]

- **Non-pollution variables included in statistical models and their measurement.** Weather variables such as temperature, humidity, and atmospheric pressure affect health and are often correlated with air pollution. For example, in a study in Birmingham, Alabama, including humidity in the statistical model reduced the apparent effect of PM_{10} on mortality, but not all studies of PM in Birmingham included humidity the their models.[102] Weather variables can also be included based on different types of measurements. Humidity, for example, can be specified as specific humidity, relative humidity, or dew point. And just as for

[99] Moolgavkar and Luebeck, "A Critical Review of the Evidence on Particulate Air Pollution and Mortality."

[100] R. D. Morris, "Airborne Particulates and Hospital Admissions for Cardiovascular Disease: A Quantitative Review of the Evidence," *Environmental Health Perspectives*, vol. 109, suppl. 4 (2001), pp. 495-500. Even if there is no real underlying PM effect, random fluctuations in the data will create both positive and negative associations in the statistical relationship between PM and mortality on different days following a PM exposure, and these random fluctuations would average out to a zero effect overall. Picking off the one day with the greatest positive association will therefore cause an overestimate of the real PM effect. As an analogy, imagine you ask five people to each toss a coin 10 times. On average, each person will get five heads in ten tosses, but the actual number of heads will vary for each set of ten tosses. Just by chance, one person might get, say, 7 or 8 heads. Imagine that many other people do the same experiment, and all of them report results only for the person that got the highest number of heads. It would then spuriously appear that tossing a coin ten times typically results in maybe 7 or 8 heads, rather than 5.

[101] Smith et al., "Regression Models for Air Pollution and Daily Mortality: Analysis of Data from Birmingham, Alabama." Also see, Switzer, "Estimating Separately Personal Exposure to Ambient and Nonambient Particulate Matter for Epidemiology and Risk Assessment: Why and How."

[102] Smith et al., "Regression Models for Air Pollution and Daily Mortality: Analysis of Data from Birmingham, Alabama," Moolgavkar et al., "Particulate Air Pollution, Sulfur Dioxide, and Daily Mortality: A Reanalysis of the Steubenville Data."

pollution itself, these weather variables can be included with a range of different lag structures. The apparent effect of PM can vary depending on which variables are included in the model and how they are measured.

- **Accounting for Trends in Mortality Unrelated to Pollution.** Many unmeasured factors, such as demographic changes, changes in health care, etc., affect mortality and show up as trends in mortality over time. Researchers use "smoothing functions" to removing potential confounding due to these trends, yet there is no standardized means to determine the "correct" smoothing function.[103] A recent study found that changing the degree of smoothing can change the estimated health effects of pollution by a factor of three or more.[104]

Modeling decisions often must be based on the judgment of the researcher, because there are frequently no definitive criteria for making a determination of what represents the "best" approach. Therefore, conclusions vary from study to study, even when different researchers use the same data sets for the same cities.[105] The differences result from different choices regarding how to set up the mathematical model that relates health outcomes to pollution and other factors. Based on the variability of results given different approaches, a recent study concluded "there are many possible interpretations of the data and no single conclusion is definitive."[106]

Threshold and Concentration Response

A key issue in air pollution epidemiology is whether there exists a threshold below which PM has no effect on health. A related issue is the concentration-response function (CRF)—the rate at which health effects increase with increasing pollution levels—above the threshold level. A number of studies have reported evidence that there is no threshold for PM health effects and that the CRF increases linearly with increasing PM levels.[107] However, critics point out that any errors in the measurement of pollution exposures will cause an *underestimate* of a threshold, should one exist, and will cause a non-linear CRF

[103] Lumley and Sheppard, "Time Series Analyses of Air Pollution and Health: Straining at Gnats and Swallowing Camels?"

[104] R. Klemm "Reanalysis of Harvard Six-City Mortality Study Replication," EPA Workshop on GAM-Related Statistical Issues in PM Epidemiology, Durham, North Carolina, November 4-6, 2002.

[105] See, for example, Smith et al., "Regression Models for Air Pollution and Daily Mortality: Analysis of Data from Birmingham, Alabama," Moolgavkar et al., "Particulate Air Pollution, Sulfur Dioxide, and Daily Mortality: A Reanalysis of the Steubenville Data," Clyde, "Model Uncertainty and Health Effect Studies for Particulate Matter," Moolgavkar and Luebeck, "A Critical Review of the Evidence on Particulate Air Pollution and Mortality," Moolgavkar, "Review of Chapter 8 of the Criteria Document for Particulate Matter (Comments Submitted to EPA)," and J. M. Samet et al., "New Problems for an Old Design: Time Series Analyses of Air Pollution and Health," *Epidemiology*, vol. 14, no. 1 (2003), pp. 11-12.

[106] Smith et al., "Regression Models for Air Pollution and Daily Mortality: Analysis of Data from Birmingham, Alabama."

[107] Recent examples include M. J. Daniels et al., "Estimating Particulate Matter-Mortality Dose-Response Curves and Threshold Levels: An Analysis of Daily Time-Series for the 20 Largest US Cities," *American Journal of Epidemiology*, vol. 152, no. 5 (2000), pp. 397-406, and J. Schwartz et al., "The Concentration-Response Relation between $PM_{2.5}$ and Daily Deaths," *Environmental Health Perspectives*, vol. 110, no. 10 (2002), pp. 1025-9.

to appear linear.[108] In addition, a number of studies have reported identifying a threshold below which PM does not appear to affect health.[109]

Harvesting

A central question in air pollution epidemiology is: To the extent that acute increases in PM cause death, does PM reduce life expectancy by only days in already-frail people or by months or years in healthy people? If the latter is the case, PM could have a large effect on public health. If the former, the health effects of PM would be far smaller.

The harvesting hypothesis centers on the idea that there is a population of already-frail individuals with an average life expectancy of only a few days, who are "pushed over the edge" by some external stress, such as pollution or hot weather. People in an already frail condition have an impaired ability to maintain a stable internal environment and this prevents them from adapting to even small changes in the external environment.[110]

A number of studies have concluded that most mortality from daily air pollution variability does not represent harvesting, but rather death is advanced by months or years.[111] However, these studies did not directly assess when deaths occurred in relation to PM levels, but inferred a lack of harvesting indirectly from the mathematical properties of the statistical model used for the analysis. In addition, once again due to the properties of the models used, deaths could be counted as due to PM increases even if the deaths *preceded* the increases in air pollution—a physically nonsensical proposition if PM is indeed causing the deaths.[112]

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[108] See, for example, Lipfert and Wyzga, "Statistical Considerations in Determining the Health Significance of Constituents of Airborne Particulate Matter."

[109] R. L. Smith et al., "Threshold Dependence of Mortality Effects for Fine and Coarse Particles in Phoenix, Arizona," *Journal of the Air and Waste Management Association*, vol. 50, no. 8 (2000), pp. 1367-79, R. L. Smith et al., "Assessing the Human Health Risk of Atmospheric Particles," *Novartis Foundation Symposium*, vol. 220 (1999), pp. 59-72; discussion 72-9, Smith et al., "Regression Models for Air Pollution and Daily Mortality: Analysis of Data from Birmingham, Alabama," Moolgavkar and Luebeck, "A Critical Review of the Evidence on Particulate Air Pollution and Mortality."

[110] R. Frank and C. Tankersley, "Air Pollution and Daily Mortality: A Hypothesis Concerning the Role of Impaired Homeostasis," *Environmental Health Perspectives*, vol. 110, no. 1 (2002), pp. 61-5.

[111] S. L. Zeger et al., "Harvesting-Resistant Estimates of Air Pollution Effects on Mortality," *Epidemiology*, vol. 10, no. 2 (1999), pp. 171-5, A. Zanobetti et al., "Generalized Additive Distributed-Lag Models: Quantifying Mortality Displacement," *Biostatistics*, vol. 1 (2000), pp. 279-92, J. Schwartz, "Harvesting and Long Term Exposure Effects in the Relation between Air Pollution and Mortality," *American Journal of Epidemiology*, vol. 151 (2000), pp. 440-48, J. Schwartz, "Is There Harvesting in the Association of Airborne Particles with Daily Deaths and Hospital Admissions?" *Epidemiology*, vol. 12, no. 1 (2001), pp. 55-61.

[112] Switzer, "A Review of Statistical Methods Used in Time-Series Epidemiologic Studies of Ambient Particulate Matter and Acute Health Effects Cited by the April 2002 EPA Draft PM Criteria Document."

Studies that have attempted to estimate directly when death occurs in relation to increases in pollution by estimating the size of this frail population have concluded that acute changes in pollution levels shorten life expectancy by a matter of days at most.[113]

The putative effects of PM based on epidemiologic results are consistent with the harvesting hypothesis. For example, if daily variations in pollution mainly affect an already-frail population, it may be that it's not so much the type of external stress that is important, but that any modest external stress would be enough to cause death. This is consistent with the finding that many different types of pollution—e.g., fine and coarse PM, various gases—appear to have effects on mortality of similar magnitude, as do changes in temperature, atmospheric pressure and other weather variables.[114] If PM and other pollutants were shortening healthy people's lives by months or years, it would be an odd coincidence if several different pollutants, each with a different intrinsic toxicity and each present at different levels in different cities, all happened to exert roughly the same effects, regardless of the pollutant or its ambient concentration.

On the other hand, if PM is actually shortening life by months or years in otherwise healthy people, biological plausibility is still an issue. Various pollutants are always present at some level in ambient air, and pollution levels vary from day to day. It is not clear why apparently healthy people would be suddenly killed on a given day by relatively low PM levels that they have experienced many times in the past.[115] The frail-population hypothesis would explain the possible lack of a threshold for the effect of PM on mortality, since changes in pollution, even at low levels, might be enough to cause death in very frail people.[116]

Responsible Components of PM

PM is composed of many chemicals, with major components including organic compounds and ammonium sulfate formed from ammonia and SO_2 emissions. PM also includes trace amounts of many other compounds, such as various metals emitted from a wide range of sources. Although some of these compounds are toxic given high enough exposures, it is not clear which might be toxic at typical ambient levels.

Sulfates appear to be an unlikely cause of PM health effects. Sulfate occurs naturally in bodily fluids, and the amount of sulfate inhaled from ambient PM is at most a tiny

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[113] Smith et al., "Assessing the Human Health Risk of Atmospheric Particles," C. J. Murray and C. R. Nelson, "State-Space Modeling of the Relationship between Air Quality and Mortality," *Journal of the Air and Waste Management Association*, vol. 50, no. 7 (2000), pp. 1075-80.

[114] F. W. Lipfert, "Unresolved Questions in Air Pollution Epidemiology, Review Comments on the U.S. Environmental Protection Agency's Air Quality Criteria for Particulate Matter, Third External Review Draft" (Annapolis, MD: Annapolis Center for Science-Based Public Policy, 2002), Stieb et al., "Meta-Analysis of Time-Series Studies of Air Pollution and Mortality: Effects of Gases and Particles and the Influence of Cause of Death, Age, and Season."

[115] Lipfert, "Unresolved Questions in Air Pollution Epidemiology, Review Comments on the U.S. Environmental Protection Agency's Air Quality Criteria for Particulate Matter, Third External Review Draft."

[116] Ibid., Frank and Tankersley, "Air Pollution and Daily Mortality: A Hypothesis Concerning the Role of Impaired Homeostasis."

fraction of the amounts that are naturally present.[117] Toxicology studies have found that ammonium sulfate inhalation has no detrimental effects on lung function or other respiratory parameters.[118] Furthermore, inhaled magnesium sulfate is used therapeutically to *reduce* airway constriction in asthmatics.[119] Although acidic aerosols, such as sulfuric acid, can have adverse effects, very high concentrations—70 $\mu g/m^3$ or more, which is many times greater than ambient levels—are necessary to induce changes in lung function, even in asthmatics.[120]

Studies using concentrated ambient PM suggest that trace metals found in PM are likely candidates for the biologically active component.[121] In a recent study, concentrated PM was "instilled"—that is, placed directly into the lungs—of human volunteers. The PM was collected from air in the Utah Valley during periods before, during, and after the temporary closure of a local steel mill. PM collected during operation of the steel mill had relatively high levels of iron, copper, zinc, vanadium, and other metals and caused lung inflammation in the volunteers, while PM from the period of steel mill closure had low metal content and provoked little or no inflammation.[122]

Although there has been little toxicology research on the organic components of PM, a few epidemiologic studies have assessed which components of PM are most strongly associated with health effects. Some of these studies have reported vehicle-related PM to be the component most associated with increased mortality.[123] However, trace metals, rather than organic or elemental carbon, might be responsible for this association.[124]

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[117] D. J. Edwards et al., "Plasma Concentrations of Inorganic Sulfate in Alzheimer's Disease," *Neurology*, vol. 43, no. 9 (1993), pp. 1837-8, D. E. Cole, "Microassay of Inorganic Sulfate in Biological Fluids by Controlled Flow Anion Chromatography," *Journal of Chromatography*, vol. 225 (1981), pp. 359-367.

[118] R. B. Schlesinger and L. C. Chen, "Comparative Biological Potency of Acidic Sulfate Aerosols: Implications for the Interpretation of Laboratory and Field Studies," *Environmental Research*, vol. 65, no. 1 (1994), pp. 69-85, J. Q. Koenig, et al., "Respiratory Effects of Inhaled Sulfuric Acid on Senior Asthmatics and Nonasthmatics," *Archives of Environmental Health*, vol. 48, no. 3 (1993), pp. 171-5. Koenig et al. used ammonium sulfate as an inert control—that is, a compound expected to have no effect on health—to compare with inhalation of sulfuric acid.

[119] L. J. Nannini, Jr. and D. Hofer, "Effect of Inhaled Magnesium Sulfate on Sodium Metabisulfite-Induced Bronchoconstriction in Asthma," *Chest*, vol. 111, no. 4 (1997), pp. 858-61.

[120] J. Q. Koenig, et al., "Respiratory Effects of Inhaled Sulfuric Acid on Senior Asthmatics and Nonasthmatics," EPA, "Air Quality Criteria for Particulate Matter, Third External Review Draft," pg. 7-27.

[121] R. S. Chapman et al., "Ambient Particulate Matter and Respiratory and Cardiovascular Illness in Adults: Particle-Borne Transition Metals and the Heart-Lung Axis," *Environmental Toxicology and Pharmacology*, vol. 4 (1997), pp. 331-8.

[122] A. J. Ghio and R. B. Devlin, "Inflammatory Lung Injury after Bronchial Instillation of Air Pollution Particles," *American Journal of Respiratory and Critical Care Medicine*, vol. 164, no. 4 (2001), pp. 704-8.

[123] See, for example, F. Laden et al., "Association of Fine Particulate Matter from Different Sources with Daily Mortality in Six U.S. Cities," *Environmental Health Perspectives*, vol. 108, no. 10 (2000), pp. 941-7, T. F. Mar et al., "Associations between Air Pollution and Mortality in Phoenix, 1995-1997," *Environmental Health Perspectives*, vol. 108, no. 4 (2000), pp. 347-53, and Hoek et al., "Daily Mortality and Air Pollution in the Netherlands."

[124] For example, the Laden et al. study used PM data collected when leaded gasoline was still in use, meaning that vehicle-related PM would have included a great deal of lead, making it much different from current vehicle-related PM composition.

Summary of Short-Term PM Effects

There is still substantial uncertainty as to the degree of increased mortality due to daily variation in PM levels. Questions remain over the degree to which confounding has been removed, the existence of a threshold, and the extent to which PM has been definitively identified as the responsible pollutant. Subjective modeling decisions appear to have a large effect on the extent to which PM appears associated with short-term health effects. To the extent changes in daily PM levels do increase mortality, the evidence suggests that PM is shortening life by no more than a few days in already-frail individuals. The recent discovery of the software problem has also called into question the validity of previous results reported in the research literature. To the extent that PM at current levels is causing harm, progressive refinements in statistical methods have tended to substantially reduce the size of the estimated PM effects.

Adequacy of EPA's Assessment of PM Health Effects

EPA's pollution standards are based on the agency's assessment of pollution risks. However, EPA's regulatory documents create an unwarranted impression of certainty regarding the overall conclusions to be drawn from PM health effects research. EPA produces reports called "criteria documents" (CD) to provide the scientific backing for its health standards. A number of researchers have pointed out that EPA's latest CD for particulate matter[125]—a report intended to be an objective and rigorous review of the health effects of PM—omits or misrepresents many studies that are critical of the view that relatively low current PM levels cause harm, and cherry picks results from the research literature that are favorable to EPA's proposed $PM_{2.5}$ standards.[126] For example, one commenter noted that of 400 studies related to PM and health published in peer-reviewed journals, 180 were not cited in the CD. Furthermore, studies omitted by EPA were more likely to have found smaller or non-existent PM health effects when compared with studies EPA chose to include in the CD.[127] This suggests that EPA has not adequately considered the weight of the evidence in setting its latest PM standards.

The Mar et al. study used a statistical technique called "factor analysis," which attempts to identify groups of variables (in this case, gaseous pollutants and the individual components of PM) that cluster together into a smaller number of underlying "factors." Each factor might represent a different major source for a given group of pollutants. For example, aluminum, silicon, calcium, and iron (all found in trace amounts in PM) fall into one factor that probably represents soil dust. Organic carbon, potassium, and bromine cluster together into a factor that probably represents vegetative burning. NOx, CO, lead, zinc, iron, manganese, elemental carbon, and organic carbon cluster into a factor that probably represents a combination of motor vehicle exhaust and road dust resuspended into air by passing vehicles. It is possible that the metals, which are mainly from resuspended road dust, are the cause of the association between this factor and mortality.

[125] EPA, "Air Quality Criteria for Particulate Matter, Third External Review Draft."

[126] Lipfert, "Review Comments on the U.S. Environmental Protection Agency's Air Quality Criteria for Particulate Matter, Third External Review Draft," Moolgavkar, "Review of Chapter 8 of the Criteria Document for Particulate Matter (Comments Submitted to EPA)."

[127] Lipfert, "Review Comments on the U.S. Environmental Protection Agency's Air Quality Criteria for Particulate Matter, Third External Review Draft."

Net Welfare Effects of PM regulations

The health effects of PM at current levels appear to be small, yet the costs of attaining the annual $PM_{2.5}$ standard will likely be quite large. Senator Jeffords's Clean Power Act or the Bush Administration's Clear Skies Initiative would add to these costs. This makes it difficult to ensure that pollution reduction measures will result in net health benefits for the people whom the regulations are intended to help. The policy problem is that pollution reduction measures involve "health-health" tradeoffs for the public.[128]

Reducing pollution may improve health. But regulations to reduce pollution increase the cost of useful goods and services, reducing families' disposable income. Because people on average use their income to make their lives safer—by buying better and safer products, more nutritious food, better medical care, and more leisure time—reducing people's disposable income reduces their health.

For example, electricity provides power for safety-enhancing services such as air conditioning. An epidemiologic study found, after controlling for confounders, that risk of death during a five-year period declined 42 percent for people who had central air conditioning in their homes, when compared with people without air conditioning.[129] Yet measures to reduce power plant emissions will increase the cost of electricity. Policymakers must assess all the effects of a regulation to ensure that the net result will be improved public health and welfare.

A number of researchers have attempted to estimate the health costs imposed by regulations. These estimates suggest that every $15 million in additional regulatory costs results in one additional induced fatality.[130] Expected health benefits of a regulation must be weighed against these health costs in order to increase the likelihood that a given regulation will provide net health benefits to the public.

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[128] Randall Lutter and John Morrall appear to be the first to use this term (see R. Lutter and J. F. Morrall, "Health-Health Analysis: A New Way to Evaluate Health and Safety Regulation," *Journal of Risk and Uncertainty*, vol. 8 (1994), pp. 43-66.

[129] E. Rogot et al., "Air-Conditioning and Mortality in Hot Weather," *American Journal of Epidemiology*, vol. 136, no. 1 (1992), pp. 106-16.

[130] R. Lutter et al., "The Cost-Per-Life-Saved Cutoff for Safety-Enhancing Regulations," *Economic Inquiry*, vol. 37, no. 4 (1999), pp. 599-608. Fifteen million dollars was their "best estimate," with a range of $10 million to $50 million.

Health-health analysis is only a partial analysis of the net welfare effects of a regulation, because such analyses currently include only mortality. Cost-benefit analyses attempt to include all costs and benefits of a regulation—not only mortality, but also morbidity (that is, disease and disability), and all the other social-welfare effects of a regulation. In this sense, health-health analysis is a weaker test of the value of a regulation than cost-benefit analysis. However, because it is a weaker test, if a regulation cannot be shown to have net health benefits in a health-health analysis, than it is very likely that the regulation in question will cause net harm to the public. Health-health analysis also has the virtue of making the net health effects of a regulation explicit to the public, while cost-benefit analysis is often perceived (inaccurately) as divorced from concerns over human welfare.

EPA did not include the negative health effects of regulatory costs when setting standards for $PM_{2.5}$.[131] EPA's Regulatory Impact Analysis (RIA) for its $PM_{2.5}$ standard also understates by a large margin the likely costs of attaining the standard. EPA estimated annual full attainment costs at $6.3 billion per year, but a more realistic estimate is at least several times greater.[132] Nevertheless, EPA estimated that full attainment of $PM_{2.5}$ standards would save 15,000 lives per year. Using a similar analysis, EPA estimates that the Clear Skies Initiative would save as many as 12,000 lives by 2020, while costing $3.7 billion annually by 2010 and $6.5 billion by 2020.[133]

If reducing particulates could save that many lives, even costs of tens of billions per year would likely be justified. However, the discussion above of $PM_{2.5}$ health effects showed that current $PM_{2.5}$ levels are probably not high enough to be causing increased deaths except at worst in a handful of locations with extremely high average $PM_{2.5}$ levels. Attaining the current $PM_{2.5}$ standard might therefore not result in any health benefits in all but a few non-attainment areas.[134]

Considering the net welfare effects of pollution-control regulations makes explicit the tradeoffs between the health benefits of lower pollution levels, and the health costs of reducing people's disposable income through imposition of regulatory costs. In the case of the annual $PM_{2.5}$ standard, the costs to the public of measures needed to achieve the standards, combined with the small health benefits that would accrue, will likely cause a net reduction in public health.[135]

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[131] However, EPA argued, and the Supreme Court agreed, that the Clean Air Act prohibits EPA from considering implementation costs when setting air quality health standards (see *Whitman v. American Trucking Associations*, 531 U.S. 457 (2001), supct.law.cornell.edu/supct/html/99-1257.ZS.html).

[132] EPA's $PM_{2.5}$ RIA included only control measures expected to cost less than $1 billion per 1 $\mu g/m^3$ reduction in annual $PM_{2.5}$ levels. But EPA's own analysis indicated that these measures would achieve only half the reductions necessary to achieve the standard, and that marginal costs per $\mu g/m^3$ reduction would rise steeply after the less expensive measures had been implemented. EPA's contractor found that costs for Philadelphia would be at least $4.3 billion per 1 $\mu g/m^3$ reduction, and that the city still might not be able to attain the standard. Based on this figure, a University of Rochester economist estimated national full-attainment costs at $55 billion per year (Stephen Huebner and Kenneth Chilton, "EPA's Case for New Ozone and Particulate Standards: Would Americans Get Their Money's Worth," Center for the Study of American Business, Washington University in St. Louis, June 1997, csab.wustl.edu/csab/CSAB%20pubs-pdf%20files/Policy%20Studies/PS139%20Huebner-Chilton.pdf).

[133] For EPA's estimates, see www.epa.gov/air/clearskies/benefits.html, www.epa.gov/air/clearskies/econ.html.

[134] For example, only 3 percent of $PM_{2.5}$ monitoring locations (10 percent of all non-attainment locations) have annual-average $PM_{2.5}$ levels greater than 20 $\mu g/m^3$. Yet, as shown earlier, epidemiologic research suggests that to the extent $PM_{2.5}$ is causing increased mortality due to long-term exposure, the threshold is somewhere above 20 $\mu g/m^3$.

[135] The high cost of attainment is at least partially due to the Clean Air Act requirement for attainment within the next 5 to 10 years. Most of the costs result from imposing new and costly requirements in advance of "natural" emission reductions that will occur anyway due to turnover of vehicle fleets and other capital stocks (see emission trends section, above).

Activists' Portrayals of PM Risks

PM and other air pollutants have been declining for decades. Current trends in vehicle-fleet turnover and already-adopted regulations for industrial sources of pollution ensure continued pollution declines in coming years. The case for long-term harm from current levels is relatively weak, while short-term changes in PM levels likely shorten life by no more than a matter of days.

Despite this relatively optimistic picture, the public's view of air pollution is just the opposite of reality. Numerous polls show most Americans believe that air pollution has been getting worse or will get worse in the future, and that air pollution is a serious threat to most people's health.[136] One reason for Americans' misperception may be a series of reports from activist groups featuring alarmist rhetoric and misleading portrayals of air pollution levels and health effects.[137]

These reports come under scary titles such as "Darkening Skies;" "Death, Disease and Dirty Power;" and "Power to Kill;" and claim that power plant PM pollution causes 30,000 deaths per year, mainly from coal-fired power plants in the eastern United States. Each of these reports sources the 30,000 deaths claim back to a study commissioned by the Clean Air Task Force, a coalition of environmental groups, and carried out by consultants from Abt Associates.[138]

The Abt study bases its PM-induced mortality estimates on $PM_{2.5}$ effects reported in the ACS cohort study. But, as shown above, the ACS results are likely spurious, suffering from confounding by non-pollution factors not accounted for in the ACS analysis. In addition, the Veterans study and the County study concluded that $PM_{2.5}$ either has no effect on long-term mortality, or that the threshold for harm is somewhere above 20 $\mu g/m^3$—well above $PM_{2.5}$ levels at 97 percent of U.S. monitoring locations. Furthermore, the areas that do have $PM_{2.5}$ greater than 20 $\mu g/m^3$ are mainly located in southern California and California's southern Central Valley, where there are no coal-fired power plants and electricity generation produces no sulfur dioxide and contributes only about 2 percent of regional NOx emissions. The evidence from toxicology studies also shows that sulfates—the portion of PM from coal-fired power plants—have no effect on health. Indeed, inhaled magnesium sulfate is used therapeutically to treat asthmatics.

Given this evidence, the Abt report and the activist reports derived from it have vastly exaggerated the health damage from current levels of PM pollution and the health effects of power plant emissions.

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[136] See, for example, ICR Media, "Survey of Air Pollution Perceptions, Final Report," www.cleanairprogress.org/research/Perceptions.pdf, and www.cleanairprogress.org/news/quorum_res_01_14_02.asp; New York League of Conservation Voters, "Key Findings of A Statewide Survey of New York State Residents on Environmental Issues," (New York, 2001), www.nylcv.org/Programs/NYCEF/NYSPoll_PDF_file.PDF; Mark Baldassare, "PPIC Statewide Survey: Special Survey on Californians and the Environment," (San Francisco, Public Policy Institute of California, 2002), www.ppic.org/publications/CalSurvey28/survey28.pdf.

[137] See, for example, Clean Air Task Force, "Power to Kill", Public Interest Research Group, "Darkening Skies", Clean Air Task Force, "Death, Disease and Dirty Power."

[138] Abt Associates, "The Particulate-Related Health Benefits of Reducing Power Plant Emissions."

Readers of these reports would also never know that PM levels have been dropping and will continue to drop. For example, the Public Interest Research Group's (PIRG) "Darkening Skies" reports that 300 power plants increased their SO_2 emissions between 1995 and 2000. Once emitted, some SO_2 gets converted into sulfate particulates through chemical reactions in the atmosphere. But PIRG never mentions that overall SO_2 emissions declined 33 percent between 1973 and 1999; that total power plant SO_2 emissions declined 29 percent from 1990 to 2000; and that federal law requires an additional 20 percent SO_2 reduction from power plants between 2000 and 2010.[139] PIRG also fails to mention that sulfate PM levels across the eastern U.S. have declined by 10 to 40 percent since the late 1980s, due to these SO_2 reductions.[140] Indeed, "Darkening Skies" contains no information at all on actual trends in pollutant emissions or actual PM levels in any community, despite the wealth of data available from hundreds of monitoring locations in populated areas around the country.

Instead of providing the public with a realistic assessment of air quality, PIRG's report misleads readers to draw conclusions grossly at odds with reality. Other activist-group reports followed similar recipes, using superficially scary, but misleading statistics, while omitting information on actual air pollution levels, trends, and risks.[141]

Policy Considerations

The analysis presented above suggests the following policy considerations and recommendations:

The epidemiologic evidence suggests the annual $PM_{2.5}$ standard should be revised upward to at least 20 $\mu g/m^3$. EPA's annual $PM_{2.5}$ standard is based mainly on the ACS study. Yet this study likely suffers from residual confounding, making its results unreliable. Other recent studies of long-term $PM_{2.5}$ exposure have found either no effect or a threshold greater than 20 $\mu g/m^3$. An annual PM standard of 20 $\mu g/m^3$ has the benefit of being stringent enough to protect public health from chronic $PM_{2.5}$ exposure, while at the same time ensuring that public health isn't harmed by diverting tens of billions per year of Americans' income to attaining an unnecessarily stringent standard. For the same reasons, the evidence does not support the Jeffords Clean Power Act, the Administration's Clear Skies Initiative, or any other costly new measures designed to further reduce PM from relatively low current levels.

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[139] R. E. Baumgardner et al., "Measurements of Rural Sulfur Dioxide and Particle Sulfate: Analysis of CASTNET Data, 1987 through 1996," *Journal of the Air and Waste Management Association*, vol. 49 (1999), pp. 1266-79, EPA, "EPA's Acid Rain Program: Results of Phase I, Outlook for Phase II."

[140] Based on EPA CASTNET data for 42 locations with data for both the late 1980s and the last few years. Data were downloaded from EPA's CASTNET data site, www.epa.gov/castnet/data.html.

[141] For a more detailed exposition of this issue, focusing on ozone air pollution, see Joel Schwartz, "A Dose of Reality on Air Pollution Exposure and Trends," *Regulation* (Summer 2003, in press).

Hardly any areas of the country exceed the EPA's daily $PM_{2.5}$ standard. The substantive case for harm from daily variation in PM at current levels is plagued by uncertainties and appears to be weaker than assumed by advocates for a more stringent standard. Progressive refinements in epidemiologic methods have resulted in smaller estimates of acute PM risks, and PM exposure more likely shortens life by days in the already-frail, rather than months or years in healthy individuals.

Even though policymakers and environmental activists have focused their PM policy efforts on power plants, sulfate is implausible as the component of PM responsible for harm. In any case, the Clean Air Act requires a 20 percent reduction in power plant SO_2 emissions between 2000 and 2010. To the extent that vehicle-related PM can cause harm at current levels, the good news is that current fleet turnover trends mean vehicle PM pollution will continue declining regardless of other policy actions. Vehicle emissions will decline at least 70 to 80 percent during the next 20 years or so, as older vehicles are scrapped and replaced by progressively lower-emitting and more durable newer models. This means that already-adopted measures have essentially mitigated PM and other air pollutants as a long-term problem. The key question for policy makers then is, to the extent some areas currently have harmful PM levels, what policies make the most sense for achieving PM reductions in the near term?

On-road emissions measurements show that a few percent of (mainly older) gasoline-powered vehicles contribute most emissions from the gasoline-powered vehicle fleet.[142] Remote sensing, an on-road emissions measurement technology, can rapidly and cheaply identify many and perhaps most of these vehicles, and their owners can be offered cash to voluntarily scrap the vehicle.[143] For example, an aggressive program could reduce gasoline-vehicle VOC emissions by at least 10 percent within a year and at a nationwide cost of no more than about $500 million.[144] While some areas of the country have small scrap programs, because there is probably no more cost effective or more rapid means for

[142] For example, when cars are ranked from dirtiest to cleanest on VOC emissions, the worst 5 percent of cars produce about 50 percent of tailpipe VOC emissions from the vehicle fleet. Likewise, when cars are ranked based on NOx, the worst 5 percent of NOx emitters produce about 35 percent of NOx from the vehicle fleet. (Based on analysis of remote sensing data for Phoenix, Chicago, and Riverside, CA, downloaded from www.feat.biochem.du.edu/light_duty_vehicles.html).

[143] Pilot programs have shown that even a relatively modest remote sensing campaign can measure a large fraction of the vehicles registered in a region. For example, a pilot program in Sacramento measured 45 percent of registered vehicles in Sacramento County with 555 "unit-days" of measurements—where one unit-day represents a single remote sensing unit operating for a day. In this case, the measurements were made by 10 units operating for about two months each. Another pilot program in Greeley, Colorado measured 70 percent of the area's fleet. (R. Klausmeier et al., "Draft Final Report - Evaluation of the California Pilot Inspection/Maintenance (I/M) Program" (Sacramento, California: California Bureau of Automotive Repair, 1995), R. Klausmeier and P. McClintock, "The Greeley Remote Sensing Pilot Program - Final Report" (Denver: Colorado Department of Public Health and Environment, 1998))

[144] There are roughly 200 million light-duty gasoline vehicles in the U.S. Assuming that half of these are in areas that need additional air pollution reductions, encouraging accelerated scrappage of 0.5 percent of them would likely cost no more than about $500 million (assuming an average cost of $1,000 per scrapped vehicle—the high end of what recent programs have offered). For an overview of issues in designing scrappage program, see Eastern Research Group, "Overview of Vehicle Scrappage Programs for Reducing In-Use Vehicle Emissions," (Austin, TX: July 2002).

achieving large air pollution reductions, this approach deserves a far more aggressive effort in areas with pollution problems.

A similar approach can be applied to diesel vehicles and equipment. However, because diesel engines last much longer than automobiles, retrofitting modern PM controls, or "repowering" older engines with new, lower-emitting ones are usually better choices than scrappage. No one has yet tried to target high-emitting diesels using remote sensing, but this may be possible as well. EPA has recently encouraged voluntary retrofit programs, while California provides funding for an incentive program to encourage public agencies and private businesses to repower or retrofit diesel vehicles and equipment.[145] Preliminary cost-effectiveness estimates suggest that diesel retrofit programs can also be much more cost effective than most other options for reducing NOx and PM pollution.[146]

Scrappage and retrofit programs would thus reduce both direct PM emissions and emissions of secondary PM precursors. Such programs have substantial advantages over blanket national regulations on power plants or new vehicles. First, they can be tailored based on the types of emission reductions most desirable in a given region. Second, they can be targeted toward the most cost effective emission reductions. Third, because they have few sunk costs, they can be easily scaled up or scaled back, depending on regional pollution-reduction needs and the availability of funding.

These programs also entail far fewer risks than either additional emission reduction requirements on power plants or on new vehicles. The latter programs result in substantial ongoing increases in energy costs and costs of new vehicles,[147] while scrappage and retrofit are one-time costs that speed the permanent removal of a large source of emissions. In addition to the direct harm these extra costs will impose on consumers, increasing the cost of new vehicles will also slow fleet turnover and its attendant pollution reductions. Nevertheless, regulatory agencies and environmental activists have emphasized additional controls on power plants and new vehicles, rather than more cost effective programs to deal with older high-polluting vehicles.

The evidence suggests that exposure to PM at current levels likely has little or no effect on mortality in most of the United States. Regardless, processes already set in

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[145] EPA, www.epa.gov/otaq/retrofit/, and California Air Resources Board, "The Carl Moyer Program Annual Status Report, March 26, 2002, www.arb.ca.gov/msprog/moyer/moyer.htm.

[146] CARB, "The Carl Moyer Program Annual Status Report," March 26, 2002, www.arb.ca.gov/msprog/moyer/2002report.pdf. The program generally does not target pollution reduction projects in order of cost effectiveness, so the cost effectiveness of retrofit program could probably be improved even further.

[147] For example, EPA estimates its "Tier II" regulation requiring substantial reductions in emissions from new gasoline vehicles starting in 2004 will cost $5.3 billion. This will make new cars more expensive, but will achieve relatively few overall emission reductions, because newer cars are already so much cleaner than the average car on the road. The federal Energy Information Administration (EIA) estimates that a 75 percent reduction in average power-plant NOx and SO_2 emissions (below levels already required under Title IV of the Clean Air Act and EPA's NOx "SIP Call" regulation) would add a few billion dollars per year to the nation's electricity bill (EPA, "Regulatory Impact Analysis: Tier 2 / Gasoline Sulfur Final Rulemaking." EIA, "Analysis of Strategies for Reducing Multiple Emissions from Power Plants: Sulfur Dioxide, Nitrogen Oxides, and Carbon Dioxide" (Washington, DC: 2000)).

motion guarantee substantial PM reductions in coming years. Additional near-term reductions in PM are probably best achieved by dealing with the stock of high-polluting older vehicles that account for a substantial portion of ambient PM levels in metropolitan areas. This flexible, more cost-effective approach is far more likely to result in net public health benefits than other proposals that are the focus of current legislative and regulatory activity and debate.

ABOUT THE AUTHOR

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