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Air Pollution and Health: Do Popular Portrayals Reflect the Scientific Evidence?

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Environmentalists, regulators, health scientists, and journalists are the main purveyors of information on air pollution health risks. Unfortunately, these groups create the appearance that harm from air pollution is much greater and more certain than suggested by the underlying evidence. The incentives in air pollution health research encourage risk exaggeration, because information purveyors depend on public fear to maintain their funding and influence. Investigative reporters are in the best position to assess how the political economy of environmental health research affects the production and portrayal of the evidence. Public debate on air pollution will continue to proceed from false premises until journalists take up this challenge.

In a nationwide survey in 2004, 85 percent of Americans rated air pollution as a "very serious" or "somewhat serious" problem, with similar results for state surveys.[1] In a recent Gallup Poll, 78 percent of Americans said they worry about air pollution "a fair amount" or "a great deal."[2] Public fear of air pollution is understandable, because most popular information about air pollution is indeed alarming.

Activist groups regularly issue reports with scary titles such as *Danger in the Air; Death, Disease and Dirty Power; Highway Health Hazards; Plagued by Pollution; and Children at Risk*.[3] Health researchers often issue alarming summaries of their research as well. Recent press-release headlines from health research institutes include "Smog May Cause Lifelong Lung Deficits," "Link Strengthened between Lung Cancer, Heart Deaths and Tiny Particles of Soot," "USC Study Shows Air Pollution May Trigger Asthma in Young Athletes," and "Traffic Exhaust Poisons Home Air."[4]

Regulators declare "code orange" and "code red" alerts on days when air pollution is predicted to exceed federal health standards. And news stories on air pollution often feature menacing headlines such as "Air Pollution's Threat Proving Worse than Believed," "Don't Breathe Deeply," "Study Finds Smog Raises Death Rate," "State's Air Is among Nation's Most Toxic," and "Asthma Risk for Children Soars with High Ozone Levels."[5]

Headlines like these might be warranted if they accurately reflected the weight of the scientific evidence. But they do not. Through exaggeration, omission of contrary evidence, and lack of context, regulators, activists, and even many health scientists misrepresent the results of air pollution health studies and the overall weight of the evidence from the research literature. They create the appearance that harm from air pollution is much greater and more certain than suggested by the underlying evidence.

Journalists are the final line of defense between the public and the proponents of air pollution health scares. Unfortunately, the majority of media air pollution health stories are sensationalized exaggerations of air pollution's risks.

Through several case studies, this essay shows that misinformation on air pollution and health is a pervasive problem. As a result, public fear of air pollution is out of all proportion to the minor risks posed by current, historically low air pollution levels.

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False Alarm on Asthma and Air Pollution

Beginning in 1993, the California Air Resources Board (CARB) funded the Children's Health Study (CHS). Researchers from the University of Southern California (USC) tracked several thousand California children living in twelve communities with air pollution ranging from near-background to the worst in the nation.

At a joint press conference in 2002, the USC researchers and CARB managers reported that children who played three or more team sports were more than three times as likely to develop asthma if they lived in the six highest-ozone communities in the study, when compared with the six lowest-ozone communities.[6] They also claimed the study's results applied to cities across the United States.

Ironically, the CHS asthma study actually showed just the opposite. While higher ozone was associated with a greater risk of developing asthma for children who played three or more team sports (8 percent of children in the study), higher ozone was associated with a 30 percent *lower* risk of developing asthma in the full sample of children in the study.[7] While this fact was discussed in a journal article on the study, it was not mentioned at the press conference.[8]

Higher levels of other pollutants, including nitrogen dioxide and particulate matter (PM_{10}), were also associated with a lower asthma risk.[9] Also mentioned in the journal article, but not at the press conference, was that when the researchers divided the twelve communities in three groups of four (rather than two groups of six), the association of ozone with increased asthma prevalence in child athletes applied only to the four communities in the highest ozone group and not to the medium-ozone group.

The assertion that the study is relevant for other parts of the country was also false. The four high-ozone areas in the study averaged 89 days per year exceeding the federal eight-hour ozone standard and 59 days per year exceeding the one-hour standard during 1994–1997, the years used to assess pollution exposure in the study.[10] No area of the United States, outside of a few parts of California, has *ever* had ozone levels this high even for a single year, much less for several years running.

In fact, by the time of its release in February 2002, the study no longer applied even in the southern California areas where it was performed. Eight-hour ozone exceedances had declined 55 percent, and one-hour exceedances had declined 78 percent in the interim. By 2002, communities that were "high-ozone" areas during the study had become "medium-ozone" areas, for which ozone had no effect on asthma risk.

At the press conference releasing the CHS asthma results, the chairman of the Air Resources Board claimed: "This study illustrates the need not to retreat but to continue pushing forward in our efforts to strengthen air pollution regulations."[11] But if anything, the CHS asthma study showed that current standards already include a large safety margin. Ozone was not associated with a change in asthma risk in the medium-ozone areas of the study. Yet these areas exceeded federal ozone standards by large margins—an average of 41 eight-hour exceedance days per year and 17 one-hour exceedances.

> Through exaggeration, omission of contrary evidence, and lack of context, regulators, activists, and even many health scientists misrepresent the results of air pollution health studies and the overall weight of the evidence from the research literature.

False information on the CHS asthma results was not limited just to CARB officials or USC scientists. Health experts from around the country misinterpreted the study's results. For example, on the day the study was released, a professor at the State University of New York at Stony Brook, who has since become the American Lung Association's medical director, claimed: "This is not just a Southern California problem. There are communities across the nation that have high ozone."[12] According to the *Houston Chronicle*, Houston asthma specialists said the study showed that "Houston [should] step up its efforts to implement a state plan to reduce ozone."[13] The director of the pediatric asthma program at the University of California at Davis claimed "Sacramento is a very high ozone area, so this [the CHS asthma study] is going to be very relevant to us."[14]

Not only were all of these nominal experts wrong about whether the study is relevant to actual ozone levels in the United States, all of them completely missed the fact that ozone and other air pollutants were associated with an overall lower risk of developing asthma.

In a recent commentary on air pollution and asthma in the *Journal of the American Medical Association*, two prominent air pollution health researchers claimed:

"Some evidence suggests that air pollution may have contributed to the increasing prevalence of asthma."[15] The "evidence" they cite is the CHS asthma study.

Journalists also often act as cheerleaders for air pollution alarmists when reporting on air pollution and health. For example, a recent editorial headline in the *Sacramento Bee* declared "Smog and Asthma: The Link—and Threat—Are Real."[16] The *Bee*'s source for this claim? Once again, the CHS asthma study.

Much Ado about Very Little

The Children's Health Study also suggests that even the highest air pollution levels in the nation are having little or no effect on children's lung development. But once again, the scientists involved in the study obscured that fact.

After following more than 1,700 children from ages ten to eighteen (years 1993 to 2001), CHS scientists reported that there was no association between ozone and lung-function growth.[17] This is despite the fact that the twelve communities in the study ranged from zero to more than 120 eight-hour ozone exceedance days per year, and zero to more than 70 one-hour ozone exceedance days per year during the study period.[18] Once again, no area outside California has ever had anywhere near this frequency of elevated ozone, even for a single year, so we can conclude that ozone is not causing any reduction in children's lung capacity. This has not stopped environmental groups from claiming otherwise. For example, in *Impacts of Ozone on Our Health*, the Carolinas Clean Air Coalition claims: "Children have a 10 percent decrease in lung function growth when they grow up in more polluted air."[19]

The Children's Health Study also suggests that fine particular matter ($PM_{2.5}$) is causing little or no long-term harm to lung growth. Unlike ozone, $PM_{2.5}$ actually was associated with a small effect on lung development. Annual-average $PM_{2.5}$ levels ranged from about 6 to 32 micrograms per cubic meter ($\mu g/m^3$) in the twelve communities in the study.[20] Across this range, $PM_{2.5}$ was associated with about a 2 percent decrease in forced expiratory volume in one second (FEV_1) and a 1.3 percent decrease in force vital capacity (FVC), both measures of lung capacity.

But even this small effect drastically inflates the apparent importance of the results. First, no location outside of the CHS communities has $PM_{2.5}$ levels anywhere near 32 $\mu g/m^3$. In fact, outside California there is not a single area with $PM_{2.5}$ above 21 $\mu g/m^3$. And by the time the study was published in 2004, even the highest $PM_{2.5}$ area in California was at 25 $\mu g/m^3$.

It is also worth noting that the children in the CHS were already ten years old when they entered the study in 1993 and had therefore been breathing the even-higher air pollutant levels extant during the 1980s in southern California. For example, Riverside averaged about 48 $\mu g/m^3$ $PM_{2.5}$ during the 1980s, or about 50 percent greater than the highest $PM_{2.5}$ level measured during the CHS years.[21] If it were really these higher 1980s $PM_{2.5}$ levels that caused the lung-function declines, then the current worst $PM_{2.5}$ in the country would be causing about a 1 percent decrease in FEV_1 and a 0.5 percent decrease in FVC. Thus, taking the CHS results at face value, ozone is having no effect on children's lung development anywhere in the United States. $PM_{2.5}$ is having virtually no effect.

Nevertheless, the USC researchers' press release on the study created an unwarranted appearance of serious harm. Titled "Smog May Cause Lifelong Lung Deficits," the press release asserted: "By age 18, the lungs of many children who grow up in smoggy areas are underdeveloped and will likely never recover."[22] The National Institutes of Health (NIH) also misled the public about the study's findings and relevance. The director of the National Institute of Environmental Health Sciences claimed the study "shows that current levels of air pollution have adverse effects on lung development in children."[23]

Furthermore, although the study is relevant only to a few areas of California with uniquely high air pollution levels, by asserting that it applies to all "smoggy areas" and to "current levels of air pollution," NIH and USC created the false impression that the study applies to much of the United States.

The scientists were able to create these false impressions, because the journal article on the study, which was published in the prestigious *New England Journal of Medicine* (*NEJM*), does not explicitly reveal the magnitude of the percentage change in children's lung capacity. Instead, readers have to be vigilant enough to realize that the percentage change can be calculated by combining information found in three different places in the article.[24] It is odd that a study whose main outcome measure is changes in lung capacity never actually states the percentage change explicitly.

The researchers reported a different outcome measure in their *NEJM* paper: the percent of children in

each community with a lung capacity of less than 80 percent of the "predicted" value for their age.[25] Between the least and most polluted communities, $PM_{2.5}$ was associated with nearly a five-fold increase in this percentage, from about 1.6 percent of children in the lowest-$PM_{2.5}$ community, up to about 7.9 percent in the highest-$PM_{2.5}$ community.

This seems like a large effect, but it is not. What is going on is that the 2 percent average decline in lung function in the highest-$PM_{2.5}$ community relative to the lowest meant a shift of some children who were at, say, 80 or 81 percent of "predicted" lung capacity for their age, down to maybe 78 or 79 percent. Because lung-capacity scores have a bell-curve distribution, and few children have low lung capacity, there are many more children slightly above 80 percent than slightly below 80 percent. A small shift in average lung-capacity scores therefore results in a large change in the fraction of children scoring below a given cutoff level.[26]

Reporting that even the highest air pollution levels in the country were associated with only a 2 percent decrease in lung capacity would not have caused much alarm. This probably explains why that number is nowhere to be found in the *NEJM* report or the press releases on it.

NIH took advantage of this omission in its press release, which begins: "Children who live in polluted communities are five times more likely to have clinically low lung function—less than 80 percent of the lung function expected for their age."[27] Note how this statement creates the appearance of a decline of more than 20 percent in average lung function by leading readers to tacitly make the incorrect assumption that all children would be at 100 percent if there were no air pollution.

This is exactly the mistake environmentalists have made in promoting the study. For example, the American Lung Association's (ALA) *State of the Air 2005* report claims the "average drop in lung function was 20 percent below what was expected for the child's age."[28] The Carolinas Clean Air Coalition made a similar error.[29]

The ALA clearly did not understand the study's results. But NIH and the USC researchers created the confusion. The editors and peer reviewers at the *New England Journal of Medicine* also bear responsibility for not requiring that its article on the study explicitly state the percentage change in lung capacity associated with air pollution.

> Scientists, regulators, and environmentalists have ignored these weaknesses and continue to make believe these spurious statistical correlations are telling us something real about the effects of low-level air pollution.

Monkey Business

A University of California at Davis press release begins "Primate Research Shows Link between Ozone Pollution, Asthma."[30] The press release goes on to claim the ozone exposures in the study "mimic the effect of exposure to occasional ozone smog—for example as it occurs in the Sacramento area."

In fact, the ozone exposures in the study were far higher than the actual ozone levels in American air—including the air in Sacramento. The monkeys were exposed to 0.5 parts per million (ppm) ozone for eight hours a day for five days in a row, followed by nine days of clean air. This cycle was repeated eight times. To give you an idea of the magnitude of these ozone exposures, during the last thirty years only one site in the U.S. has ever exceeded 0.5 ppm ozone for even one hour, and that happened in 1976. Today, the worst site in the United States never reaches even 0.25 ppm for one hour, and the average site never reaches 0.11 ppm.

Despite the real-world irrelevance of this study, environmental activists cite it to support claims that ozone is causing permanent lung damage in people. For example, under the headline "Lung Development of Young Monkeys Drastically Changed when Exposed to Ozone Pollution," the American Lung Association concludes, "This study presents data suggesting that the changes caused by ozone pollution are long-lasting, and maybe even permanent."[31]

Some reporters also failed to compare ozone levels in the study to real-world ozone levels. For example, according to the *Modesto Bee*, "Monkeys were exposed to air contaminated with ozone, mimicking the smog in the [Central] valley."[32] But even more nuanced stories still took an alarmist tack. For example, the *Sacramento Bee* explicitly compared ozone levels in the Sacramento region with the far higher ozone levels used in the study.[33] But you have to go halfway into the 1,100-word story to find this information. The story's headline—"Study Suggests Asthma Culprit; Young

Lungs Exposed to Ozone Seem More Prone to Problems with Development"—leaves no doubt that readers are supposed to conclude that ozone is causing Americans to develop asthma.

Of Mice and Men

By far the most serious health claim about air pollution is that it kills tens of thousands of Americans each year, mainly due to exposure to $PM_{2.5}$. There is no question that high levels of air pollution can kill. About 4,000 Londoners died during the infamous five-day "London Fog" 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.[34]

However, current fears center on whether today's comparatively low levels of air pollution are also deadly. An embarrassment for proponents of low-level air pollution as a cause of death is that the evidence is almost solely circumstantial, being based on statistical studies reporting small correlations between long- or short-term air pollution levels and risk of dying. These "observational" studies are not based on randomized trials, but on non-random data that inherently suffer from confounding by non-pollution factors with much larger effects on health than the purported effects of air pollution.

Observational studies could be taken more seriously if they were supported by evidence from randomized, controlled studies that eliminate the possibility of confounding by non-pollution factors. Such studies cannot, of course, be done with people, but they can be done with animals. However, researchers have been unable to kill animals with air pollution at levels anywhere near as low as the levels found in ambient air. As a recent review of particulate matter toxicology concluded:

> It remains the case that no form of ambient PM—other than viruses, bacteria, and biochemical antigens—has been shown, experimentally or clinically, to cause disease or death at concentrations remotely close to U.S. ambient levels.[35]

This seemingly changed in December 2005 when the *Journal of the American Medical Association* (*JAMA*) published the results of a study that claimed $PM_{2.5}$ at current ambient levels is increasing Americans' risk of developing heart disease. The study exposed mice to 85 $\mu g/m^3$ of $PM_{2.5}$ concentrated from ambient air for six hours per day for six months, or about one-fourth of a typical mouse life span.[36]

Mice fed a high-fat diet and exposed to $PM_{2.5}$ had more than a 50 percent greater rate of atherosclerosis (as measured by arterial plaque area) and other signs of heart disease, when compared with a control group that was fed a high-fat diet, but not exposed to $PM_{2.5}$. $PM_{2.5}$ was associated with greater atherosclerosis in mice on a low-fat diet as well, but the effect was not statistically significant.

NIH highlighted the study with a press release that begins: "Test results with laboratory mice show a direct cause-and-effect link between exposure to fine particle air pollution and the development of atherosclerosis . . . [The study] may explain why people who live in highly polluted areas have a higher risk of heart disease."[37] The study caused a minor media sensation, with both journalists and health experts claiming the study provides strong evidence that $PM_{2.5}$ is causing serious harm to human beings.[38]

Despite the enthusiastic reception, there is much less here than meets the eye. The mice used in the study were genetically engineered in ways that make them unrepresentative of even real-world mice, much less of humans. The mice were designed to lack the gene for apolipoprotein E (ApoE), a key substance for fat and cholesterol metabolism. As a result, these ApoE "knockout" mice have blood cholesterol levels 5 to 6 times greater than normal mice when fed regular rat chow. ApoE knockout mice have 14 times the cholesterol of normal mice when both are fed a high-fat diet.[39]

These are stupendous cholesterol levels. For comparison, medical authorities define "high cholesterol" as a serum cholesterol level greater than 240 milligrams per deciliter (mg/dl), which is about 20 percent greater than the average cholesterol level in American men.[40] Only one in 50 American men exceeds 1.5 times the U.S. average, and only one in 500 exceeds twice the average.[41]

The very reason for using such grossly unrealistic mice to study $PM_{2.5}$ is that $PM_{2.5}$ does not kill regular mice or other animals at PM concentrations relevant to real-world human exposures. For that matter, $PM_{2.5}$ did not actually kill the high-cholesterol mice in the study either.

NIH downplayed the vast gulf between the genetically engineered mice and normal mice, stating only that they were "genetically programmed to develop atherosclerosis at a higher-than-normal rate." This is a bit

like doing a study on people who weigh 500 pounds and referring to them merely as "overweight."

If you build a house out of cards, you would expect even a gentle breeze to knock it down. But this does not tell you much about the ability of a real house to withstand a gentle breeze. Likewise, if you design an artificial mouse that cannot regulate its fat or cholesterol levels, it is not surprising that even a minor environmental insult can cause it some health problems. But this does not tell you much about the effects of low-level air pollution levels on regular mice or on people.

Unfortunately, news articles on the study failed to provide the context that would show that study has little real-world relevance. A Nexis search turned up ten news reports on the study. Seven did not even mention that the mice had been genetically engineered, leaving the impression that real-world $PM_{2.5}$ levels caused heart disease in normal mice.

Three other news outlets followed NIH's lead, creating the impression that the mice in the study were merely analogous to people with a higher-than-average risk of heart disease. For example, according to the *Los Angeles Times*, the mice were "bred to be susceptible to developing heart disease."[42]

NIH and the study authors also misled reporters about the relevance of the $PM_{2.5}$ doses to real-world $PM_{2.5}$ levels. According to NIH, "The fine particle [$PM_{2.5}$] concentrations used in the study were well within the range of concentrations found in the air around major metropolitan areas." The press release also quotes one of the study's authors saying that "the average exposure over the course of the study was 15 micrograms per cubic meter, which is typical of the particle concentrations that urban area residents would be exposed to, and well below the federal air quality standard of 65 $\mu g/m^3$ over a 24-hour period."[43]

In fact, the $PM_{2.5}$ levels in the study were nothing like real-world $PM_{2.5}$ levels. The mice were exposed to $PM_{2.5}$ at 85 $\mu g/m^3$ for six hours in a row during five days of each week, and filtered air the rest of the time. Over the six-month study period, this does indeed average out to about 15 $\mu g/m^3$, the level of the federal $PM_{2.5}$ annual standard. But in the real world, areas that average 15 $\mu g/m^3$ of $PM_{2.5}$ over a year rarely approach short-term $PM_{2.5}$ levels of 85 $\mu g/m^3$.

For example, in the mouse study, the mice spent the equivalent of 1,560 hours per year breathing 85 $\mu g/m^3$ $PM_{2.5}$ (30 hours per week times 52 weeks per year). In contrast, Modesto California averaged 16 $\mu g/m^3$ of $PM_{2.5}$ over the past year, but spent only 80 hours at 85 $\mu g/m^3$ or above.[44] Furthermore, 40 percent of those high-$PM_{2.5}$ hours occurred between 11 p.m. and 6 a.m., when most people are in bed. There were only 420 hours when Modesto exceeded even 50 $\mu g/m^3$ of $PM_{2.5}$.

Even areas with the highest $PM_{2.5}$ levels in the country have far fewer hours of high $PM_{2.5}$ than were used in the mouse study. For example, Riverside California averaged 27 $\mu g/m^3$ $PM_{2.5}$ over the past year, but had only 135 hours at or above 85 $\mu g/m^3$, and 1,055 hours above 50 $\mu g/m^3$.

Health effects depend not only on the average dose, but on the acute dose. For example, you could take 2 aspirins 4 times per day, or you could take 8 all at once each day. Either way, your average dose is 8 aspirins per day. But you are more likely to suffer ill effects if you take the aspirins all at once. The mice received an analogously unrealistic daily $PM_{2.5}$ exposure. NIH and the scientists involved in the study then created the false appearance that this unrealistic exposure schedule has some relevance to the real world.

There is nothing wrong with the *JAMA* mouse study in principle. It shows that when you take a mouse specially designed to have unrealistically stupendous cholesterol levels, feed it a high-fat diet, and repeatedly expose it to unrealistically high acute levels of $PM_{2.5}$, that $PM_{2.5}$ increases the extent of heart disease. The problem arose when the study's proponents claimed that this has something to do with $PM_{2.5}$ risks faced by human beings.

You can now find a summary of the study on NIH's website. Its title? "Particulate Air Pollution and a High Fat Diet: A Potentially Deadly Combination."[45]

Sins of Omission

At the March meeting of the California Air Resources Board, staff members gave a detailed presentation on Jerrett et al. (2005)—a new epidemiological study of the Los Angeles region that reported a stronger link between $PM_{2.5}$ and mortality than suggested in previous research regulators have used to support tougher $PM_{2.5}$ standards.[46] What CARB's staff did not tell its board is that right around the same time that Jerrett et al. was published, another study of $PM_{2.5}$ risks in California by Enstrom (2005) concluded that $PM_{2.5}$ was having no effect on mortality.[47] Several California papers, including the *Los Angeles Times*, covered the alarming findings

of Jerrett et al. But none covered the benign results reported by Enstrom.

This is a typical pattern. Studies that report harm from air pollution receive a great deal of attention from regulators, environmentalists, and journalists. Studies finding no harm from air pollution are ignored. As a result, claims of harm from air pollution appear more consistent and robust than suggested by the actual weight of the evidence.

The American Lung Association's website includes an area called Medical Journal Watch, which summarizes hundreds of air pollution health studies.[48] But the site omits studies that do not report any harm from air pollution. For example, the site does not include any studies by Fred Lipfert, Suresh Moolgavkar, Richard Smith, Gary Koop, William Keatinge, or James Enstrom—all of whom have provided evidence against a connection between low-level air pollution and risk of death.[49]

The ALA also excludes specific studies and portions of studies that fail to find any harm from air pollution. For example, Medical Journal Watch does not mention Gong et al. (2003) and Holgate et al. (2003), which found little or no adverse health effects in human volunteers who breathed high levels of $PM_{2.5}$ and diesel soot, respectively.[50] The ALA does summarize the CHS findings on children's lung capacity discussed earlier, but does not mention that the study found that even the highest ozone levels in the country had no effect on lung growth.

Three studies have used CHS data to assess whether ozone is associated with increases in school absences. One study reported an increase.[51] Two reported no effect.[52] The ALA mentions only the first study on Medical Journal Watch. CARB likewise cites only the first study in its review of California's ozone standard.[53]

Coal-fired power plants have been one of environmentalists' premier targets during the last several years. In reports such as *Danger in the Air; Death, Disease and Dirty Power; Power to Kill; Children at Risk*; and many more, environmental groups claim that particulate pollution from power plants is killing thousands of Americans each year.[54] The Bush administration, a constant target of environmental groups for supposedly "gutting" power plant pollution requirements, last year adopted the Clean Air Interstate Rule (CAIR).[55] CAIR requires that power plants reduce their sulfur dioxide emissions by more than 70 percent below current levels.[56] Some sulfur dioxide is converted to ammonium sulfate in the atmosphere, and this is the main form of $PM_{2.5}$ from power plants. EPA claims these $PM_{2.5}$ reductions will prevent 17,000 premature deaths each year.[57]

Environmental groups want to increase support for ever more stringent regulations, maintain and enhance their control over other people's lives, and bring in the donations that support their activism. Regulators want to show the success of their efforts to reduce air pollution, but they also want to justify the need to preserve or expand their powers and budgets.

There is just one problem: ammonium sulfate is not toxic, even at levels many times those ever found in ambient air.[58] In fact, ammonium sulfate is used as an inert control—that is, a compound not expected to have any health effects—in studies of the health effects of acidic aerosols.[59] If ammonium sulfate is not toxic, then the campaign against $PM_{2.5}$ from power plants is based on a false premise.

Last year CARB adopted a tougher ozone standard for California.[60] To justify the tougher standard, CARB prepared a detailed report summarizing ozone health effects research. The report analyzes hundreds of health studies in nearly 1,000 pages, but fails to mention a study reporting that *higher* ozone was associated with a *lower* rate of hospital visits in California's Central Valley.[61] CARB was certainly aware of the existence of this study, because CARB funded and published it. EPA also failed to mention the study in its latest review of the federal ozone standard.[62]

EPA based its annual $PM_{2.5}$ standard mainly on the American Cancer Society (ACS) study, which followed more than 500,000 Americans in fifty cities from 1982 to 1989 and looked for correlations between $PM_{2.5}$ levels and risk of death.[63] The most recent ACS report covered the period from 1982 to 1998 and reported that each 10 $\mu g/m^3$ increase in long-term $PM_{2.5}$ levels is associated with a 4 percent increase in risk of death.[64]

The validity of epidemiological studies, such as the ACS study, depends on the assumption that correlations between air pollution and health outcomes represent genuine causal relationships. The implicit assumption is that after researchers have controlled for non-pollution

health factors like income or smoking, any residual correlation between air pollution and health represents a genuine causal linkage. Experience has shown that this assumption is false.

For example, a reanalysis of the ACS data showed that the apparent $PM_{2.5}$-mortality link was spurious. According to sensitivity analyses of the ACS data, $PM_{2.5}$ apparently kills men, but not women; those with no more than a high school degree, but not those with at least some college; and those who said they were moderately active, but not the very active or the sedentary.[65] Results like these are biologically implausible and suggest a failure to adequately control for confounding by non-pollution factors.

When migration rates into and out of various cities over time were added to the statistical model relating $PM_{2.5}$ and risk of death, the apparent effect of $PM_{2.5}$ disappeared.[66] Cities that lost population during the 1980s—Midwest "rust belt" cities—also had higher $PM_{2.5}$ levels. People left these cities, which were in economic decline, in search of work in more economically dynamic parts of the country. But people who work and have the wherewithal to migrate also tend to be healthier than the average person. Hence, what appeared to be an effect of $PM_{2.5}$ was actually the result of differential migration. Migration was just one of several confounding factors that diminished or erased the apparent harm from $PM_{2.5}$, but that were not accounted for by the ACS researchers.

This problem of spurious air pollution risk estimates is not limited to the ACS study, but is endemic to air pollution epidemiology and to epidemiology in general.[67] Nevertheless, scientists, regulators, and environmentalists have ignored these weaknesses and continue to make believe these spurious statistical correlations are telling us something real about the effects of low-level air pollution.

The Politics of Air Pollution Health Science

Most public information on air pollution and health comes from environmental activists, regulators, and health researchers. As these case studies show, their claims of harm from current, historically low air pollution levels are at best exaggerations and at worst fabrications. The result is unwarranted public fear, and continued support for ever more costly regulatory requirements that deliver little or no benefit in exchange for their high costs.

Regulators, environmentalists, and scientists enjoy substantial credibility with the public and the press. But like other interest groups, their goals often do not coincide with the interests of the vast majority of Americans. Environmental groups want to increase support for ever more stringent regulations, maintain and enhance their control over other people's lives, and bring in the donations that support their activism. Regulators want to show the success of their efforts to reduce air pollution, but they also want to justify the need to preserve or expand their powers and budgets. Maintaining a climate of crisis and pessimism meets these institutional goals, but at the expense of encouraging people to exaggerate the risks they face.

While it is not surprising that activists and regulators exaggerate air pollution risks, they would not be taken as seriously without scientific authority to back them up. The credibility of science and scientists flows from the power of scientific methods to uncover truths about the world, and from the perceived objectivity of scientists themselves. As the case studies above show, trust in scientific authority is often misplaced.

Scientific and medical research does have checks and balances that are absent from more explicitly political endeavors. Environmental health research nevertheless suffers from its own set of pressures that militate against evenhanded inquiry and dispassionate analysis and presentation of evidence. Studies that report harm from air pollution are more likely to be published than studies that do not. Regulatory agencies, whose power and budgets depend on the perception that air pollution is a serious health problem, are also major funders of the research intended to demonstrate the severity of the problem. Scientists who believe air pollution is a serious health threat and who report larger health effects are more likely to attract research funding. It is not a big leap to conclude that there is a great deal of selection bias in who does environmental health research, what questions they ask, and how they report their results.

Journalists should be acting as a check on air pollution misinformation, but they are not. Media outlets face their own pressures to sensationalize stories. Good news does not sell newspapers or attract viewers. As a result, journalists and editors are more likely to cover studies claiming harm from air pollution, and to pass along these claims with little or no critical review.

True, few journalists have the expertise to evaluate the technical merits of specific studies. But continuing

to rely on scientific authority will only perpetuate the problem of risk exaggeration. Among the major providers of public information on environmental risks, investigative reporters are in the best position to assess how the political economy of environmental health research affects the production and portrayal of scientific evidence. It would be a breath of fresh air if journalists and editors took up this challenge.

AEI editor Scott R. Palmer worked with Mr. Schwartz to edit and produce this Environmental Policy Outlook.

Notes

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5. T. Avril, "Air Pollution's Threat Proving Worse than Believed," *Philadelphia Inquirer*, November 17, 2004; M. Cone, "State's Air Is among Nation's Most Toxic," *Los Angeles Times*, March 22, 2006, available at www.latimes.com/news/printedition/la-me-cancer22mar22,1,7087336.story; M. Cone, "Study Finds Smog Raises Death Rate," *Los Angeles Times*, November 17, 2004; T. Freemantle, "Asthma Risk for Children Soars with High Ozone Levels—Study," *Houston Chronicle*, February 1, 2002; and T. Webber, "Don't Breathe Deeply," *Indianapolis Star*, June 23, 2005.

6. California Air Resources Board, "Study Links Air Pollution and Asthma," news release, January 31, 2002, available at www.arb.ca.gov/newsrel/nr013102.htm.

7. The risk of developing asthma was 30 percent lower based on one-hour ozone levels and was statistically significant. Asthma risk was 20 percent lower based on eight-hour ozone levels and was just a hair short of statistical significance. (The top of the 95 percent confidence interval for relative risk was 1.0. Anything less than that would have been statistically significant.)

8. The journal article is R. McConnell, K. T. Berhane, F. Gilliland et al., "Asthma in Exercising Children Exposed to Ozone: A Cohort Study," *Lancet* 359 (2002): 386–91.

9. Once again the risk was 20 percent lower and was just barely short of statistical significance.

10. Pollution monitoring data from the Children's Health Study were provided by CARB's staff.

11. California Air Resources Board, "Study Links Air Pollution and Asthma," news release, January 31, 2002, available at www.arb.ca.gov/newsrel/nr013102.htm.

12. Dr. Norman Edelman, quoted in S. Borenstein, "Air Pollution Is a Cause of Asthma, Study Contends," *Philadelphia Inquirer*, February 1, 2002.

13. T. Freemantle, "Asthma Risk for Children Soars with High Ozone Levels—Study."

14. In fact, even the worst areas of Sacramento never average more than a few days per year exceeding the one-hour ozone standard and 20 or so days per year exceeding the eight-hour standard—ozone levels typical of the "medium-ozone" CHS communities, in which there was no relationship between air pollution and asthma risk. Dr. Jesse Joad, quoted in C. Bowman, "Asthma's Toll: A New Study Links Children's Sports Activities in Smoggy Areas to the Illness," *Sacramento Bee*, February 1, 2002.

15. G. D. Thurston and D. V. Bates, "Air Pollution as an Underappreciated Cause of Asthma Symptoms," *Journal of the American Medical Association* 290 (2003): 1915–17.

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17. W. J. Gauderman, E. Avol, F. Gilliland et al., "The Effect of Air Pollution on Lung Development from 10 to 18 Years of Age," *New England Journal of Medicine* 351 (2004): 1057–67.

18. The CHS study set up special-purpose monitors to measure pollution levels in the communities where the study was performed. CARB staff provided data from these monitors.

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20. Based on research by CARB staff, these values have been adjusted upward by 13.6 percent to make them comparable with $PM_{2.5}$ levels determined by the Federal Reference Method, which has been used nationwide since 1999 for determining compliance with federal $PM_{2.5}$ standards. See N. Motallebi, J. Taylor, A. Clinton, B. E. Croes et al., "Particulate Matter in California: Part 1—Intercomparison of Several $PM_{2.5}$, $PM_{10\text{-}2.5}$, and PM_{10} Monitoring Networks," *Journal of the Air & Waste Management Association* 53 (2003): 1509–16.

21. Based on IPN data for Riverside collected in the early 1980s, and $PM_{2.5}$ data collected by CARB in 1988 and 1989 and retrieved from CARB's 2006 Air Pollution Data CD, www.arb.ca.gov/aqd/aqdcd/aqdcd.htm. Once again, I have corrected these values for the change in measurement methods.

22. Di Rado, "Smog May Cause Lifelong Lung Deficits."

23. Dr. Kenneth Olden, quoted in NIH, "New Research Shows Air Pollution Can Reduce Children's Lung Function," news release, September 9, 2004, www.nih.gov/news/pr/sep2004/niehs-08a.htm.

24. Here's how: First, note from table 3 of the *NEJM* article that $PM_{2.5}$ was associated with a 79.7 milliliter (ml) reduction in FEV_1 between the least and most polluted community. Then from table 2, note that at eighteen years of age average FEV_1 was 3,332 ml for girls and 4,464 ml for boys. Given that there were 876 girls and 883 boys in the study (p. 1,059, column 1), the weighted average FEV_1 for the study population was 3,900 ml. The percentage decline is then 79.7/3,900 = 0.02 or 2 percent. A similar calculation can be done to show that the average decline in FVC was 1.3 percent. Gauderman, Avol, Gilliland et al., "The Effect of Air Pollution on Lung Development from 10 to 18 Years of Age."

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