

The Health Effects of Air Pollution

Separating Science and Propaganda

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EXECUTIVE SUMMARY

Americans are alarmed about air pollution, and no wonder. Most of the information they receive about air pollution is alarming. Activist groups issue reports with scary titles such as *Danger in the Air*; *Death, Disease and Dirty Power*; or *Children at Risk*. News stories on air pollution often feature alarming headlines, such as the recent *Observer* story “Traffic is Choking Charlotte’s air.”

These portrayals of air pollution, and the fear they instill, might be warranted if they accurately reflected the health risks of current, historically low air pollution levels. But they do not. Through cherry-picking, exaggeration, and sometimes outright fabrication, environmental activists have created public fear of air pollution out of all proportion to the actual risks suggested by the underlying health studies. Regulators, journalists, and even health professionals also frequently paint a misleadingly pessimistic portrait of air pollution’s health toll.

Air pollutants of all kinds in North Carolina and the United States in general are at their lowest levels since nationwide measurements began back in the 1970s. The weight of the evidence from a wide range of health studies suggests that these low levels of air pollution are at worst a minor health concern.

Asthma provides a signal example of how conventional wisdom on air pollution and health is often the opposite of reality. Asthma prevalence has doubled in the United States at the same time that air pollution of all kinds has sharply declined. Air pollution is therefore not a plausible cause of rising asthma. A government sponsored study that followed thousands of children in California during the 1990s reported that higher ozone, particulate matter, and other air pollutants were associated with a lower risk of developing asthma. Counties in North Carolina with higher ozone levels have lower asthma hospitalization rates.

Despite the evidence, activists continue to create false scares about air pollution and asthma. For example, according to the Carolinas Clean Air Coalition, “1/3 1/2 of all asthma in North Carolina is due to air pollution.”

The California study of children and asthma also showed that even air pollution in southern California, which is by far the highest in the country, is having little or no effect on children’s lung development. The study reported that even living in areas that exceed federal ozone standards more than 100 days per year had no effect on children’s lung capacity.

Fine particulate matter PM_{2.5} at levels more than twice the federal standard was associated with only a 1 to 2 percent decrease in lung capacity. Even the worst PM_{2.5} pollution in North Carolina barely exceeds the federal standard. Thus, neither ozone nor PM_{2.5} is harming lung development of North Carolina’s children. Despite this evidence, CCAC wants to maintain a climate of fear, no matter how unwarranted. In a recent op-ed, CCAC claimed “children who grow up in areas as polluted as the Charlotte region are losing up to 20 percent of their lung function permanently.”

Attaining federal ozone and PM_{2.5} standards will cost tens to hundreds of billions of dollars per year, nationwide. These costs are ultimately paid by people in the form of higher prices, lower wages, and reduced choices. We all have many needs and aspirations and insufficient resources with which to fulfill them. Spending more on air quality means less money to spend on everything else that’s important to us, including health care, housing, food, and education, as well as measures that address larger and more certain health and safety risks. We are giving up much to fund our massive air pollution regulatory system, and getting little in return.

INTRODUCTION

Americans are alarmed about air pollution, and no wonder. Most of the information they receive about air pollution is alarming. Activist groups issue reports with scary titles such as *Danger in the Air*; *Death, Disease and Dirty Power*; or *Children at Risk*.¹ Air pollution regulators declare “code orange” and “code red” alerts on days when air pollution is predicted to exceed federal health standards. News stories on air pollution often feature alarming headlines, such as the recent *Charlotte Observer* story “Traffic is Choking Charlotte’s air.”²

These portrayals of air pollution, and the fear they instill, might be warranted if they accurately reflected the health risks of current, historically low air pollution levels. But they do not. Through cherry-picking, exaggeration, and sometimes outright fabrication, environmental activists have created public fear of air pollution out of all proportion to the actual risks suggested by the underlying health studies. Regulators, journalists, and even health professionals also frequently paint a misleadingly pessimistic portrait of air pollution’s health toll.

As this paper will show, air pollution affects far fewer people, far less often, and with far less severity than environmentalists and other trusted sources have led people to believe. It isn’t that air pollution can’t be harmful. But as toxicologists like to say, “the dose makes the poison.”

Air pollutants of all kinds in North Carolina and the United States in general are at their lowest levels since measurements nationwide began back in the 1970s. The weight of the evidence from a wide range of health studies suggests that these low levels of air pollution are at worst a minor health concern.

DOES AIR POLLUTION CAUSE ASTHMA?

Asthma provides a signal example of how conventional wisdom on air pollution and

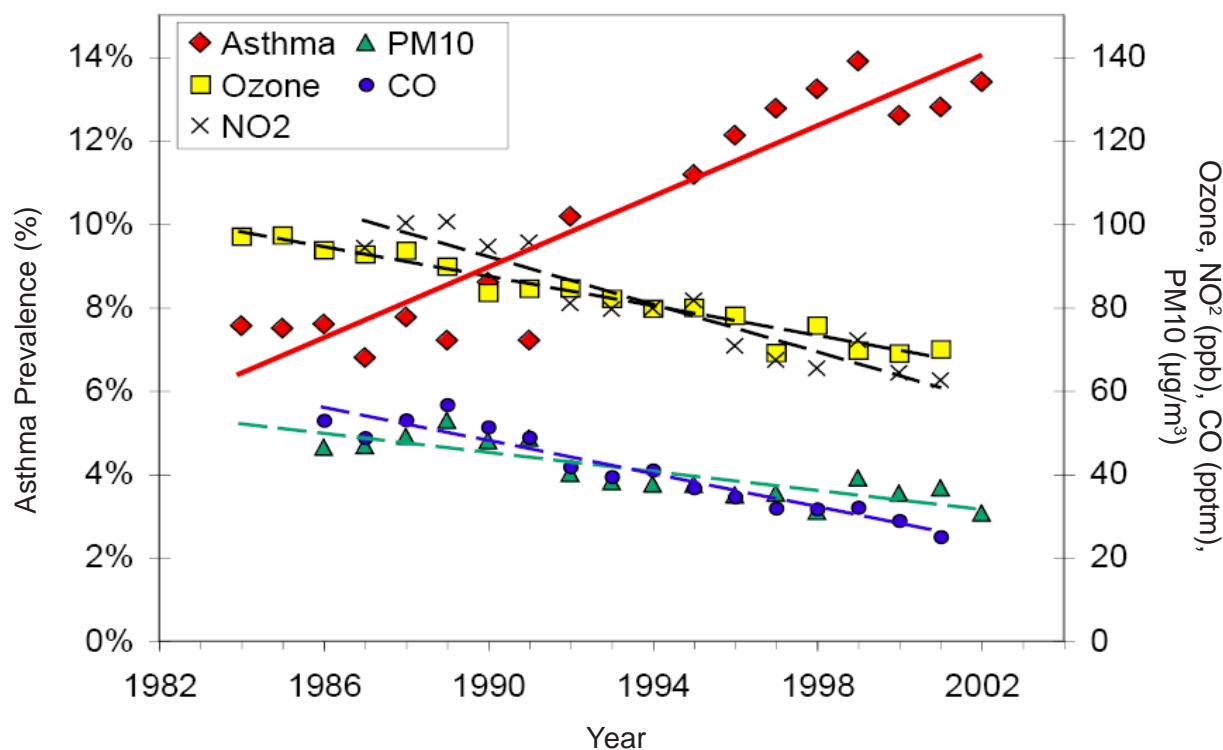
health is often the opposite of reality. According to the Centers for Disease Control, the prevalence of asthma in the U.S. rose 75 percent from 1980 to 1996, and nearly doubled for children. Prevalence may have leveled off since then.³ Could air pollution be the cause? Not likely. Asthma prevalence rose at the same time that air pollution of all kinds declined. North Carolina does not have long term measurements of asthma prevalence, but many other states do. Figure 1 (next page) displays trends in asthma and various air pollutants for California. The trends are similar for all other pollutants measured by California regulators, including fine particulate matter (PM_{2.5}),⁴ benzene, 1-3-butadiene, benzo(a)pyrene,

Air pollutants of all kinds in North Carolina are at their lowest levels since measurements began back in the 1970s. The weight of the evidence suggests that these low levels of air pollution are at worst a minor health concern.

perchloroethylene, xylene, lead, and many more.⁵ In all cases air pollution has been declining while asthma has been rising. Data from other states tell the same story — declining air pollution, rising asthma.

Despite the implausibility of air pollution as a cause of asthma, regulators and health experts have even turned a study that found air pollution to be associated with a *lower* overall risk of developing asthma into a key piece of evidence in support of an air pollution-asthma link. Beginning in 1993 the California Air Resources Board (CARB) funded the Children’s Health Study (CHS). Performed by researchers from the University of Southern California (USC), the CHS tracked several thousand California children living in 12 communities with widely varying air pollution levels, including areas of southern California with the highest air pollution levels in the country.

Figure 1. Trend in Asthma Prevalence vs. Trends in Air Pollution in California



Notes: CO = carbon monoxide, PM_{10} = airborne particulate matter under 10 micrometers in diameter, NO_2 = nitrogen dioxide; ppb = parts per billion; pptm = parts per ten million; $\mu g/m^3$ = micrograms per cubic meter.

Sources: Asthma prevalence data were provided by the California Department of Health Services. Air pollution data were extracted from the California Air Resources Board's 2003 Air Pollution Data CD. The latest edition of this CD is available at <http://www.arb.ca.gov/aqd/aqcd/aqcd.htm>.

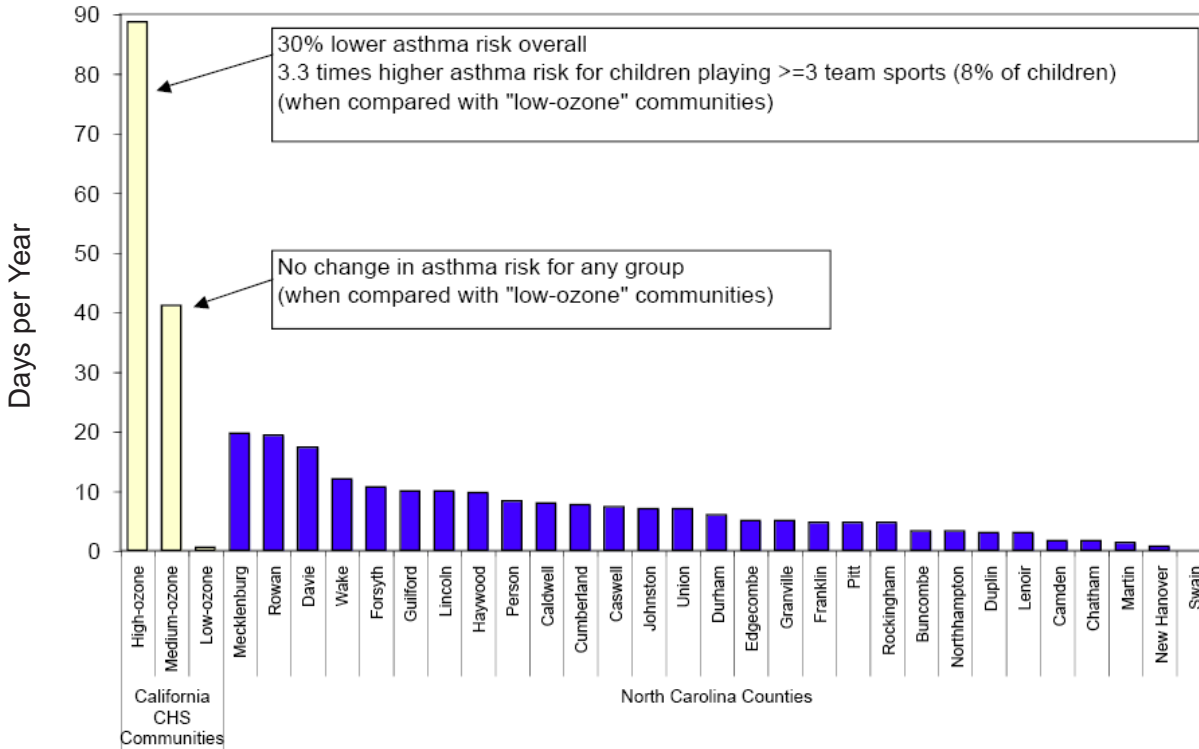
At a joint press conference in 2002, the USC researchers and CARB managers reported that children who played three or more team sports were more than three times as likely to develop asthma if they lived in high ozone communities in the study, when compared with low ozone communities.⁶ The study became the most widely cited evidence that air pollution is causing children to develop asthma and that air pollution is a major cause of rising asthma prevalence.

Ironically, the CHS asthma study actually showed just the opposite. Unmentioned at the press conference was that 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 asthma in the full sample of children in the study.⁷ Furthermore, higher levels of other pollutants, including nitrogen dioxide and particulate matter, were also associated with a *lower* asthma risk in all children. Unfortunately, the many journalists who covered the study reported only what the researchers and regulators told them, rather than what the study actually found.⁸

In a recent commentary on air pollution and asthma in the *Journal of the American Medical Association*, two prominent air pollution health researchers stated "Evidence exists that air pollution may have contributed to the increasing prevalence of asthma."⁹ The evidence they cite is the CHS asthma

Figure 2. Days per Year Exceeding the 8-hour Ozone Standard in California Children's Health Study Communities Compared with the Worst Location in Each North Carolina County



Notes: The 12 Children's Health Study (CHS) communities were ranked from worst to best and then divided into three groups of four communities each. Ozone levels during 1994-97 were then averaged for each group of four communities. These are the same groupings used in the CHS asthma study published in the Lancet. North Carolina ozone data are based upon the average number of exceedance days per year during 1999-2001 at the worst location in each county

Source: CHS data were provided by the staff of the California Air Resources Board. North Carolina ozone data were downloaded from EPA at <http://www.epa.gov/ttn/airs/airsaqs/detaildata/downloadaqsdata.htm>.

study the one that found that higher air pollution was associated with a lower risk of developing asthma.

And these researchers aren't the only ones to misinterpret the results of the CHS asthma study. 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 (ALA) medical director, claimed "This is not just a Southern California problem. There are communities across the nation that have high ozone."¹⁰

He was wrong on both counts. The CHS asthma study was based on ozone levels from 1994-97 in 12 California communi-

ties. The change in asthma risk (higher risk for children playing 3 or more team sports; lower risk for everyone else) was observed only in the four communities with the highest ozone relative to the four lowest ozone communities. These four communities averaged 89 days per year exceeding the 8-hour ozone standard during 1994-97.¹¹ The four "medium" ozone areas averaged 41 ozone exceedance days per year and had no change in asthma risk, either overall or for just the children playing three or more team sports.¹²

No area outside California has ever had ozone levels as high as the CHS high-ozone areas. In fact, by the time the study was

released in February 2002, it no longer applied even in the southern California areas where it was performed. During 1999-2001, the four “high-ozone” CHS areas averaged 40 8-hour exceedance days per year—the same as the “medium-ozone” areas, for which there was no change in asthma risk.

Figure 2 preceding page compares ozone levels in the 12 CHS communities during 1994-97 with ozone levels North Carolina counties during 1999-2001 (the three most recent years before the study was released early in 2002). For California, the graph shows the average number of ozone exceedance days per year for the each of the three groups of communities

Air pollution is not a plausible cause of asthma. Nevertheless, many media and activist reports and even some prominent medical researchers have created the impression that air pollution is a major cause of asthma.

high, medium, and low ozone. For each North Carolina county, the graph shows the number of 8-hour ozone exceedance days per year at the worst location in the county. Note that even the worst areas of North Carolina don’t come close to even the medium-ozone areas of the CHS, much less the high ozone areas.¹³

ALA’s medical director wasn’t the only one providing false information about the CHS asthma study. At the press conference releasing the study’s results, the USC researchers who performed the study and the CARB regulators who sponsored it also claimed the study’s results apply to pollution levels all around the United States.

Air pollution—at least the wide range of air pollutants that regulators measure and control, and that environmentalists sound alarms about—is not a plausible cause of asthma.¹⁴ Nevertheless, many media and activist reports and even some prominent medical researchers have cre-

ated the impression that air pollution is a major cause of asthma.

For example, according to the Carolinas Clean Air Coalition (CCAC), a Charlotte-based environmental group, “1/3 1/2 of all asthma in North Carolina is due to air pollution.”¹⁵ The CCAC provides no source for this ridiculous claim. The CCAC also claims “children with increased ozone exposure have 3.3 times the risk of developing asthma.”¹⁶

In other words, the CCAC takes a result from the Children’s Health Study that applies to 8 percent of children living in areas that average 89 8-hour ozone exceedance days per year, and applies it to all children in North Carolina—a state where no area averages more than about 20 8-hour exceedances days per year. The CCAC also completely missed the fact that the Children’s Health Study actually reported that higher ozone was overall associated with a lower risk of developing asthma. This is just one among many egregious examples of activists providing false information about the relationship between air pollution and asthma.¹⁷

DOES AIR POLLUTION EXACERBATE PRE-EXISTING LUNG DISEASE?

While air pollution is not plausible as a cause of asthma, air pollution can exacerbate pre-existing respiratory diseases. Yet even here, the effects of air pollution have been overstated in popular accounts when compared with the weight of the evidence. For example, EPA estimates that even substantial ozone reductions will result in tiny health improvements. In a recent study published in the journal *Environmental Health Perspectives*, EPA scientists estimated that reducing nationwide ozone from levels during 2002, which had by far the highest ozone levels of the last six years, down to the federal 8-hour standard would reduce asthma emergency room visits by 0.04

percent, respiratory hospital admissions by 0.07 percent, and premature mortality by 0.03 percent.¹⁸

The California Air Resources Board (CARB) recently adopted an ozone standard for California that is much tougher than the federal standard, requiring ozone to be reduced to near or even below background levels across the state.¹⁹ Despite the fact that parts of California have much higher ozone levels than the rest of the country, CARB predicts that reducing ozone will result in little health improvement. For example, based on CARB's estimates, going from ozone levels during 2001-2003 down to attainment of CARB's standard — in effect an elimination of all human caused ozone in the state — would reduce emergency room visits for asthma by 0.35 percent, respiratory-related hospital admissions by 0.23 percent, and premature mortality by 0.05 percent.²⁰

Even these benefits are exaggerated, because CARB ignored contrary evidence when generating its benefit estimates. For example, researchers from Kaiser Permanente studied the relationship between air pollution and emergency room visits and hospitalizations in California's Central Valley, and reported that higher ozone was associated with a statistically significant *decrease* in serious health effects, such as hospital admissions.²¹ CARB omitted this study from its estimate of the ostensible benefits of a tougher ozone standard.²² CARB must have been aware of the study, because CARB funded and published it. This selective use of evidence creates the impression that air pollution's effects are larger and more certain than suggested by the overall weight of the evidence.²³

The pattern of hospital visits for asthma also suggests ozone can't be a significant factor in respiratory exacerbations. Emergency room visits and hospitalizations for asthma are lowest during July and August,

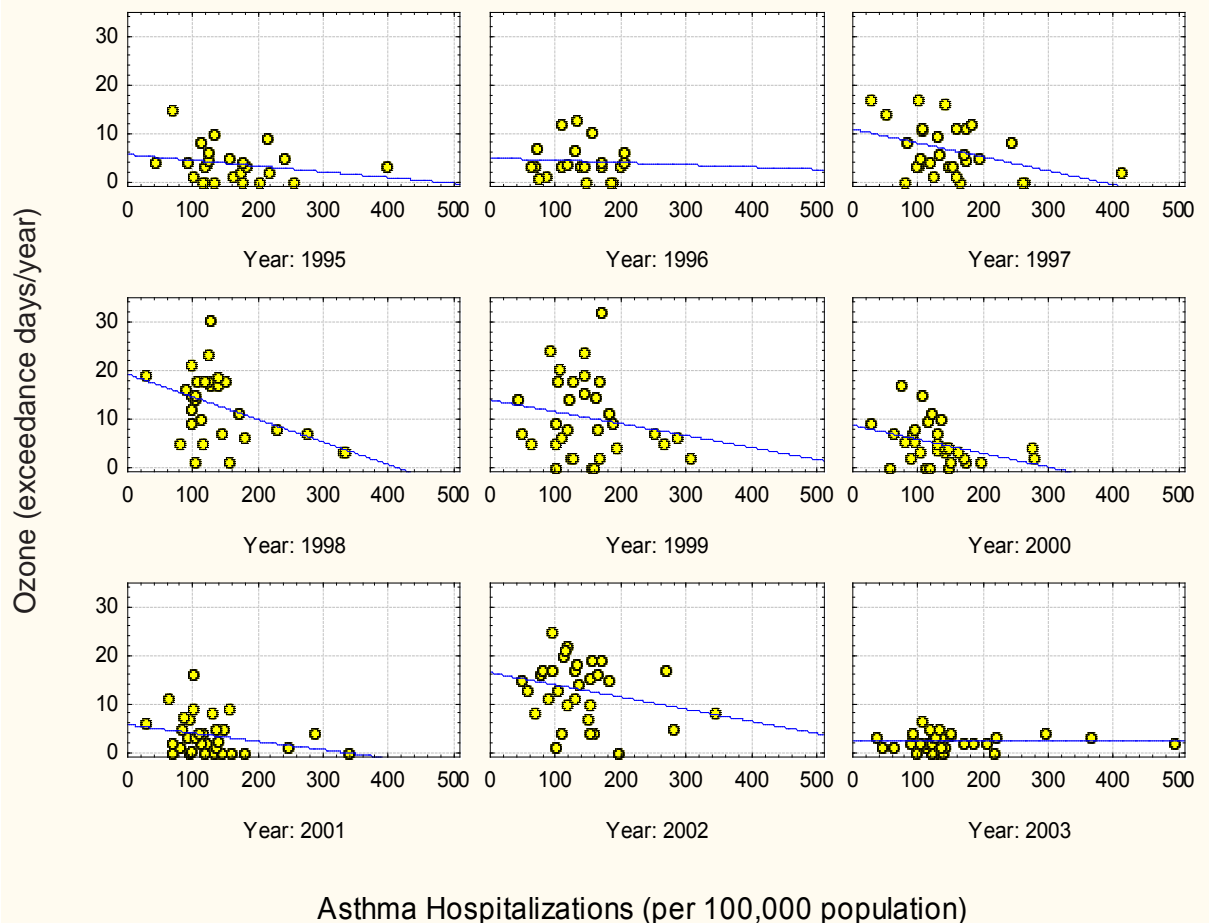
when ozone levels are at their highest.²⁴ For example, in North Carolina, counties with the *lowest* ozone levels have the *highest* rate of asthma hospitalizations. This is shown in Figure 3. Each graph represents an individual year and each point represents a North Carolina county. The vertical axis gives the number of 8-hour ozone exceedance days in that year. For counties with more than one ozone monitoring site, the ozone value is an average of all sites in the county. The horizontal axis gives the number of asthma hospitalizations per 100,000 people. The lines through the data points are linear regression lines. Note that counties with the lowest ozone have the highest asthma hospitalization rates.

LONG-TERM EFFECTS OF AIR POLLUTION

The estimates above address only short term effects of ozone. But the Children's Health Study suggests that ozone is having little effect on long-term health as well. In addition to asthma, the CHS assessed the relationship between air pollution and growth in children's lung-function.²⁵ After following more than 1,700 children from age 10 to 18 (years 1993 to 2001), the study reported that there was no association between ozone and lung function growth. This is despite the fact that the 12 communities in the study ranged from zero to more than 120 8-hour ozone exceedance days per year, and zero to more than 70 1-hour ozone exceedance days²⁶ per year during the study period.

No area outside California has anywhere near this frequency of elevated ozone, even for a single year, much less for several years running. For example, no area of North Carolina has ever had more than 16 1-hour ozone exceedance days in a year — that was Charlotte back in 1978. Since 1990, most of the state has had zero 1-hour ozone exceedance days per year and no site has ever had more than 5. The story is

Figure 3. Asthma Hospitalization Rate vs. Ozone Level for N.C. Counties



Notes: Ozone exceedance days are based on the 8-hour ozone standard.

Sources: Ozone data were downloaded from EPA at www.epa.gov/ttn/airs/airsaqs/detaildata/downloaddaqsdata.htm. Asthma hospitalization data were provided by the North Carolina State Center for Health Statistics.

similar under the new 8-hour ozone standard. The worst location in North Carolina averaged 6 exceedance days per year during 2003-2005.²⁷ In 1998, one of the worst years for ozone in North Carolina, the worst location in the state had 43 8-hour ozone exceedance days, and the average location had 15.²⁸

If 70 or 120 ozone exceedance days per year doesn't reduce kids' lung capacity in California, then North Carolina's far lower ozone levels certainly won't be causing harm either. Nevertheless, in its pamphlet on ozone's health effects, the Carolina's

Clean Air Coalition claims "Children have a 10% decrease in lung function growth when they grow up in more polluted air."²⁹

The Children's Health Study also suggests that PM_{2.5} is causing little long term harm. 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 (µg/m³) in the 12 communities in the study.³⁰ Across this range, PM_{2.5} was associated with about a 2 percent decrease in forced expiratory volume in one second (FEV₁), and a 1.3 percent reduction in full

vital capacity (FVC). Both tests are standard tests of lung function.³¹

But even this drastically inflates the apparent importance of the results, because no location outside of the CHS communities has PM_{2.5} levels anywhere near 32 µg/m³. In fact, even the worst area in the U.S. averaged 25 µg/m³ for 2002-2004. There also didn't appear to be any decrease in lung function until average PM_{2.5} levels exceeded about 15 µg/m³, which is the current level of the federal annual PM_{2.5} standard.³² But 87 percent of the nation's monitoring locations are already below 15 µg/m³. The worst location in North Carolina averaged 15.4 µg/m³ for 2002-04 and only two locations were above 15 µg/m³.

It is also worth noting that the children in the CHS were already 10 years old when they entered the study, and had therefore been breathing the even higher air pollutant levels extant during the 1980s in southern California. For example, the Riverside area averaged nearly 50 µg/m³ PM_{2.5} during the early 1980s.³³ If it was these higher 1980s pollution levels that caused the lung function declines, then the harm from current air pollution levels is even smaller than the already tiny effect reported in the CHS lung-function study.

Thus, taking the CHS results at face value, ozone is having no effect on children's lung development anywhere in the U.S. PM_{2.5} is having no effect in the vast majority of the U.S., including North Carolina. Even in areas that have the highest PM_{2.5} levels in the country, the effect on lung function is at worst about a one percent decrease.

Despite finding little effect of air pollution on children's lung growth, the USC researchers' press release on the study created the 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."³⁴ The National Institutes of Health also misled the public about the study's findings and relevance. In the NIH press release, 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..."³⁵

Both press releases created the impression that air pollution was associated with large decreases in lung function. In fact, the decrease was small, even in the most polluted areas. Furthermore, by referring to "smoggy areas" and "current levels of air pollution" the press releases created the false impression that the study is relevant

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for many areas of the United States. But in fact, even the tiny decreases in lung function apply only to a few areas in California with uniquely high air pollution levels. And even in those few areas, the study applies to pollution levels from at least a decade ago, and not to present pollution levels, which are much lower than levels during the study.

Activists likewise create a misleading impression of widespread, serious harm from current levels of air pollution. For example, in a recent *Charlotte Observer* column, Nancy Bryant of the Carolinas Clean Air Coalition claimed "Medical studies show that children who grow up in areas as polluted as the Charlotte region are losing up to 20 percent of their lung function – permanently."³⁶

I asked Ms. Bryant if she could provide the research evidence to back up this claim.³⁷ She sent me the NIH press release discussed above.

DOES AIR POLLUTION KILL?

Death is by far the most serious among potential harms from air pollution, and 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.³⁸

The question today is whether current, far lower levels of air pollution can also be deadly. EPA’s PM_{2.5} standards are based on the assumption that PM_{2.5} at current levels is killing tens of thousands of Americans each year, due to both long term exposures and the acute effects of daily PM fluctuations.³⁹

The apparent effect of PM_{2.5} was actually “caused” by healthier people moving away from areas of the country that were in economic decline, rather than from a change in any individual’s health status due to PM exposure.

EPA based its annual PM_{2.5} standard mainly on the American Cancer Society (ACS) cohort study. The ACS study followed more than 500,000 Americans in dozens of cities from 1982 to 1998.⁴⁰ In their most recent report, the ACS researchers concluded that each 10 µg/m³ increase in long term PM_{2.5} levels is associated with a 4 percent increase in risk of death.⁴¹

However, inspection of the detailed results of the ACS study suggest that PM isn’t increasing people’s risk of death. For example, the ACS study reported that 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. These results are biologically implausible and suggests problems with the

researchers’ statistical model, rather than a real cause-effect relationship.

Reanalysis of the ACS data has also shown that considering additional factors in the statistical analysis of the data can make the apparent PM_{2.5} effect disappear. For example, when migration rates into and out of cities was added to the statistical model relating PM_{2.5} and premature death, the apparent effect of PM_{2.5} declined by two thirds and became statistically insignificant.⁴²

Cities that lost population during the 1980s — Midwest “rust belt” cities that were in economic decline — also had higher average PM_{2.5} levels. People who work and have the wherewithal to migrate are healthier than the average person. These people left Midwest cities in disproportionate numbers, seeking jobs in more economically dynamic parts of the country. The people who remained behind were less healthy on average, and therefore more likely to die. Thus, the apparent effect of PM_{2.5} was actually “caused” by healthier people moving away from areas of the country that were in economic decline, rather than from a change in any individual’s health status due to PM exposure. The Harvard Six Cities study, another cohort study cited in support of PM mortality claims, suffers from similar problems.⁴³

Regulators and environmentalists have also ignored another major study that reported no association between long term PM_{2.5} levels and mortality in a cohort of 50,000 male veterans with high blood pressure — a group that should have been *more* susceptible than the average person to any pollution-related health effects.⁴⁴

Studies of the short-term health effects of daily fluctuations in air pollution levels likewise suffer from a number of difficulties that create the appearance of an association between low level air pollution and mortality where none may in fact exist.

One key problem is publication bias — the tendency for researchers and journal editors to selectively publish studies that find an air pollution health association rather than studies that fail to find such an association.⁴⁵ Furthermore, in published studies there is a tendency to screen several ways of analyzing the data, but then report the analyses that result in the largest and most statistically significant associations between air pollution and health — an effect known as model-selection bias. As a recent review of air pollution epidemiology studies concluded,

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. Because the possible mechanisms of action and their latencies are uncertain, the biologically correct models are unknown. This model selection problem is exacerbated by the common practice of screening multiple analyses and then selectively reporting only a few important results.⁴⁶

Studies of the effect of publication bias have shown that it can reduce the apparent association between daily air pollution levels and mortality by as much as 70 percent.⁴⁷ After accounting for model selection bias, a recent study concluded that the air pollution mortality association drops to zero.⁴⁸

Studies of the mortality risk of air pollution are not like drug trials, where volunteers are randomly assigned to treatment and control groups in order to isolate the real effects of the prospective drug. Instead, ethics and practicality require that researchers use non random observa-

tional data and try to statistically tease out the putative effects of air pollution from all other confounding factors that could affect health. As shown above, the results of these statistical studies must be taken with a large grain of salt. Experience with hormone replacement therapy provides additional evidence of how relying on observational epidemiology studies can lead to seriously mistaken conclusions.

Based on observational epidemiological studies of hormone replacement therapy (HRT), researchers concluded that not being on HRT increases a woman's risk of heart disease by a factor of 2.⁴⁹ An influential meta analysis of these studies, published in 1991, helped make HRT one of the most prescribed therapies in the United States.⁵⁰ But more recently, randomized

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controlled trials, which eliminate the possibility of confounding by unobserved factors that affect health, showed that HRT does not reduce heart disease risk and might even increase risk.

Thus, in the HRT case, even a 100 percent increase in risk based on epidemiological studies turned out to be spurious once all confounding effects were genuinely controlled for by doing a randomized, controlled trial. The putative risks that air pollution studies are attempting to pick out are tiny by comparison — at most a few tenths of a percent in the short term studies and a few percent in the long term studies. Furthermore, the effects of air pollution need to be separated out from a much larger array of potential confounding factors than in the case of the HRT studies. Indeed, a number of epidemiologists have suggested that epidemiological studies

are inherently unreliable for assessing the existence of such small risks.⁵¹

Given the unreliability of epidemiological studies in cases where the magnitude of the potential risk is small, it is also important to note that controlled toxicological studies with animals and human volunteers do not find evidence that air pollution can cause disease or death at concentrations anywhere near as low as the levels found in ambient air in the United States.⁵² A recent review of particulate matter 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 US ambient levels. This lack of demonstration is not for lack of trying: hundreds of researchers, in the US and elsewhere, have for years been experimenting with various forms of pollution derived PM, and none has found clear evidence of significant disease or death at relevant airborne concentrations.⁵³

Despite the conclusion above, in December 2005 the *Journal of the American Medical Association* published the results of a study that claimed even relatively low current levels of PM_{2.5} might be increasing Americans' risk of heart disease. The study exposed mice to 85 µg/m³ of PM_{2.5} drawn from ambient air for 6 hours per day for 6 months, or about one fourth of a typical mouse life span.⁵⁴ Mice fed a high fat diet had more signs of heart disease if exposed to PM_{2.5}, when compared with an unexposed control group.

The study caused a minor media sensation, with both journalists and health experts claiming the study provides proof that particulate pollution is a significant

risk factor in human heart disease.⁵⁵ But what none of the reporters or health experts mentioned is that the mice in the study had been genetically engineered to have blood cholesterol levels 5 to 6 times greater than normal mice, and 14 times normal when fed a high fat diet.⁵⁶

These are stupendous cholesterol levels. For comparison, doctors consider a person to have medically high cholesterol if his cholesterol level is more than 20 percent greater than the U.S. male average of 200 milligrams per deciliter. Only about one in-50 American men has a cholesterol level more than 50 percent above the U.S. average. And only about one-in-500 has cholesterol greater than twice the U.S. average.⁵⁷ Indeed, the very reason for using these unrealistic mice to study PM_{2.5}, is that PM_{2.5} does not kill regular mice or other animals even at PM concentrations many times greater than even the highest levels found in the United States.

REGULATORY COSTS AND AMERICANS' HEALTH

None of the discussion above would matter if we could reduce air pollution for free. But reducing air pollution is costly. Attaining the federal 8-hour ozone and annual PM_{2.5} standards will cost tens to hundreds of billions of dollars per year.⁵⁸ These costs are ultimately paid by people in the form of higher prices, lower wages, and reduced choices.⁵⁹ We all have many needs and aspirations and insufficient resources with which to fulfill them. Spending more on air quality means spending less on other things that improve our health, safety, and welfare.

Higher incomes are associated with improved health, because people spend a portion of each additional dollar of income on things that directly or indirectly improve health and safety, such as better medical care, more crashworthy cars, and more nutritious food.⁶⁰ People made poorer

by the costs of regulations do fewer of these things and are less healthy as a result. Risk researchers estimate that every \$17 million in regulatory costs induces one additional statistical death.⁶¹ Thus, regulations are not pure risk reduction measures, but instead inevitably impose tradeoffs between the health benefits of the regulation and the harm from the regulation's income-reducing costs. The costs of attaining EPA's current ozone and PM_{2.5} standards will likely be more than a thousand dollars per year for each American household. EPA is now in the process of tightening these standards, which will increase costs still further. For these huge expenditures we will at best eliminate a tiny fraction all disease and disability.

Even if we could somehow convince ourselves that additional air pollution reductions would confer net benefits, focusing on air pollution would still be a foolish policy, because other measures would provide far greater health benefits per dollar invested. Based on an assessment of more than 500 life-saving measures in four categories—environmental pollution reduction, workplace safety, injury prevention, and medical care—researchers at the Harvard School of Public Health concluded that environmental measures saved by far the fewest years of life per dollar invested.⁶²

We could glibly say that we should undertake all available risk-reduction measures and save as many lives as possible. But this begs the question. If we lived in a world of infinite resources and omniscience about the full consequences of our actions, then we would of course undertake literally all health and safety measures available. But in such a world there would be no politics or policy debates over environmental regulations or over anything else. Politics and policy debates exist exactly because resources and knowledge are scarce and insufficient to satisfy all our needs and

aspirations. Maximizing human welfare requires targeting these scarce resources in ways that generate the greatest health and welfare improvements per dollar invested. Spending money on air pollution means choosing to save far fewer lives than if the same amount of money is spent in other ways.

One might argue that talking about other ways to reduce risk is irrelevant, because it is not as if money is sitting around waiting to be spent on risk reductions and air pollution is just one of many choices. We can choose to reduce air pollution or not, but if we choose not to, this does not mean the government will fund some other risk-reduction measure(s). This reasoning implicitly assumes that only publicly determined risk-reduction priorities and expenditures are legitimate. But if people aren't

Attaining the federal 8-hour ozone and annual PM_{2.5} standards will cost tens to hundreds of billions of dollars per year. Spending more on air quality means spending less on other things that improve our health, safety, and welfare.

forced to spend money to attain EPA's standards, they will have more money to spend as they see fit. People will spend these funds to improve their health, welfare, and quality of life as they define it. As a result, they will be better off than if they had been forced to spend the money on air pollution reductions that deliver tiny benefits compared to the costs imposed.

GETTING REAL ON AIR POLLUTION AND HEALTH

Most public information on air pollution and health comes from environmental activists, regulators, and health researchers. As we've seen, most of their claims of harm from air pollution are great exaggerations or even outright fabrications. The result is unwarranted public fear and continued sup

port for counterproductive regulations.

Regulators, environmentalists, and scientists no doubt appear to be more credible sources of objective information when compared with, say, politicians or industry lobbyists. But, like other interest groups, the goals of these groups 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

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in the donations that support their activism. While regulators want to show the success of their efforts to reduce air pollution, 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.

Scientific and medical research nominally has more checks and balances, but environmental health research suffers from its own set of pressures. Studies that report harm from air pollution are more likely to

be published and to receive press coverage than studies that do not. Regulatory agencies, whose existence depends on the perception that air pollution is a serious health problem, are also major funders of the research intended to demonstrate the seriousness of the problem. Researchers who report larger health effects are probably also more likely to attract additional research funding. Scientists who choose a career in air pollution health research are probably more likely to hold an environmentalist ideology and to believe that air pollution is a serious problem. Indeed, many environmental health researchers have explicitly associated themselves with environmental groups and causes.⁶³

Journalists should be acting as a check on air pollution misinformation, but they are not. Media outlets face their own pressures to sensationalize stories. It is a journalistic truism that good news doesn't sell newspapers or attract viewers. "Airplane lands safely" is not news; "Airplane crashes" is. So it is not surprising that most news coverage of air quality — like environmental news generally — accentuates the negative and downplays the positive. Yet if journalists continue to be unable or unwilling to improve environmental reporting, Americans are likely to remain misinformed and unnecessarily afraid.

NOTES

1. Clean Air Task Force, *Death, Disease and Dirty Power: Mortality and Health Damage Due to Air Pollution from Power Plants* (Boston: October 2000), <http://www.cleartheair.org/fact/mortality/mortalitylowres.pdf>; Physicians for Social Responsibility, *Children at Risk: How Air Pollution from Power Plants Threatens the Health of America's Children* (Boston: May 2002), http://www.cleartheair.org/fact/children/children_at_risk.pdf; PIRG, *Danger in the Air* (Washington, DC: August 2003).
2. B. Henderson, "Traffic Is Choking Charlotte's Air," *Charlotte Observer*, August 7, 2005.
3. Asthma prevalence trends are estimated from the Centers for Disease Control's (CDC) annual National Health Interview Survey (NHIS). The CDC changed its asthma survey questions in 1997, preventing comparison with data collected up to 1996. Between 1997 and 2000, the CDC stopped asking people whether they currently had asthma. However, in 1997 CDC began asking people who had ever been diagnosed with asthma whether they had had an attack in the past 12 months. In 2001, CDC began once again to ask people whether they currently had asthma, but with a slightly different question than pre-1997 surveys. Based on these data, the prevalence of asthma attacks leveled off from 1997-2003, while the prevalence of asthma declined from 2001-2003. American Lung Association, *Trends in Asthma Morbidity and Mortality* (Washington, DC: May 2005), <http://www.lungusa.org/atf/cf/%07B7A8D42C2-FCCA-4604-8ADE-7F5D5E762256%07D/ASTHMA1.PDF>; D. M. Mannino, D. M. Noma, L. J. Akinbami *et al.*, "Surveillance for Asthma — United States, 1980-1999," *Morbidity and Mortality Weekly Report* 51 (SS01) (2002): 1-13.
4. PM_{2.5} is shorthand for airborne soot and dust up to 2.5 micrometers in diameter. One micrometer is one-millionth of a meter, or one-25,000th of an inch.
5. Trends in these and other pollutants were determined from monitoring data extracted from the California Air Resources Board's 2005 Air Pollution Data CD. The latest edition of the CD available at <http://www.arb.ca.gov/aqd/aqcdcd/aqcdcd.htm>.
6. For CARB's press release, see California Air Resources Board, "Study Links Air Pollution and Asthma," January 31, 2002, <http://www.arb.ca.gov/newsrel/nr013102.htm>.
7. This result is discussed in the peer-reviewed journal article the researchers published on the study. R. McConnell, K. T. Berhane, F. Gilliland *et al.*, "Asthma in Exercising Children Exposed to Ozone: A Cohort Study," *Lancet* 359 (2002): 386-91.
8. See, for example, W. Booth, "Study: Pollution May Cause Asthma; Illness Affects 9 Million U.S. Children," *Washington Post*, February 1, 2002, A1; C. Bowman, "Asthma's Toll: A New Study Links Children's Sports Activities in Smoggy Areas to the Illness.," *Sacramento Bee*, February 1, 2002, A1; M. Enge, "Study Links Pollution to Asthma in Children; Active Kids in Smoggy Areas at More Risk, Researchers Say," *San Jose Mercury News*, February 1, 2002, 21A; T. Freemantle, "Asthma Risk for Children Soars with High Ozone Levels - Study," *Houston Chronicle*, February 1, 2002, A1.
9. G. D. Thurston and D. V. Bates, "Air Pollution as an Underappreciated Cause of Asthma Symptoms," *Journal of the American Medical Association* 290 (2003): 1915-7.
10. Dr. Norman Edelman, quoted in S. Borenstein, "Air Pollution Is a Cause of Asthma, Study Contends," *Philadelphia Inquirer*, February 1, 2002, A04.
11. The 8-hour ozone standard is the current federal standard for ozone. The standard is exceeded on a given day if ozone during any consecutive 8-hour period averages more than 0.085 parts per million ppm .
12. These asthma risk change are relative for the four "low-ozone" communities, which averaged 0.6 8-hour exceedance days per year.
13. Ozone levels were higher in North Carolina in 2002, but not by much. Rowan County had the worst ozone in the state in 2002, with 28 8-hour ozone exceedance days — still well below CHS "medium" ozone areas, which average 40 exceedance days. During the last three years, North Carolina has experienced its lowest ozone levels ever, with even the worst location in the state averaging only six ozone exceedance days per year.
14. More recently, the CHS researchers have sliced the data in a different way to argue that air pollution is a large risk factor for asthma. For a critique of this more recent study, see J. Schwartz, "Asthma and Air Pollution," *Tech Central Station*, September 26, 2005, <http://www.tcsdaily.com/article.aspx?id=092605E>. The original study is J. Gauderman, E. Avol, F. Lurmann *et al.*, "Childhood Asthma and Exposure to Traffic and Nitrogen Dioxide," *Epidemiology* 16 (2005).
15. Carolinas Clean Air Coalition, *Impacts of Ozone*

on *Our Health* (Charlotte, NC: undated), http://003af56.netsolhost.com/air_basics_ozone_impact.htm.

16. As with all of the health claims on its web site, the CCAC does not cite any sources for this claim, but it clearly comes from the California CHS asthma study.

17. Among many other examples, see, for example, *Fresno Bee*, “Asthma in the Valley; More Research Is Needed into a Disease That Runs Rampant Here,” *Fresno Bee*, October 4, 2004; NRDC, “EPA Set to Launch New Study on Causes of Asthma,” October 31, 2002, www.nrdc.org/bushrecord/health_air.asp#1157; *Sacramento Bee*, “Smog and Asthma: The Link — and Threat — Are Real,” *Sacramento Bee*, May 6, 2003, B6; R. Sanchez, “In Calif., A Crackling Controversy over Smog; Illnesses Drive Push to Ban Fireplaces,” *Washington Post*, February 16, 2003, A1; D. S. Stanley, “Stop the Spread of Asthma by Cleaning up Our Air,” *Fresno Bee*, August 7, 2004, B9; Surface Transportation Policy Project, *Clearing the Air* (Washington, DC: August 2003).

18. This analysis assumes that there are no health benefits from further reductions of ozone once the standard is achieved. However, attaining the ozone standard requires reducing ozone below the standard on the worst day at the worst location in a given region. Within any given region, ozone does not exceed the standard on most days in most locations. Nevertheless, the measures necessary to attain the standard on the worst day at the worst location would also reduce ozone on other days and other locations. As a result, most of the reduction in ozone exposure occurs on days and locations in which ozone already complies with the standard. If benefits continue to accrue when ozone is reduced below the federal 8-hour standard, then the benefits of attaining the federal 8-hour standard would be several times greater — about an 0.2 percent reduction in asthma ER visits, an 0.35 percent reduction in respiratory hospital admissions, and an 0.15 percent reduction in premature deaths. B. J. Hubbell, A. Hallberg, D. R. McCubbin *et al.*, “Health-Related Benefits of Attaining the 8-Hr Ozone Standard,” *Environmental Health Perspectives* 113 (2005): 73-82.

19. California Air Resources Board, *Review of the California Ambient Air Quality Standard for Ozone* (Sacramento: March 2005), <http://www.arb.ca.gov/research/aaqs/ozone-rs/ozone-final/ozone-final.htm>.

20. J. Schwartz, *Rethinking the California Air Resources Board's Ozone Standards* (Washington, DC: American Enterprise Institute, September 2005), http://www.aei.org/doclib/20050912_Schwartzwhitepaper.pdf.

This assumes that benefits continue to accrue only until ozone levels are reduced down to the 0.070 ppm standard. If benefits continue to accrue for ozone levels below the standard, then the percentage reduction in total health effects would be about 1.8 percent for asthma ER visits and 1.2 percent for respiratory hospital admissions and 0.3 percent for premature deaths.

21. S. F. van den Eeden, C. P. Quesenberry, J. Shan *et al.*, *Particulate Air Pollution and Morbidity in the California Central Valley: A High Particulate Pollution Region* (Sacramento: CARB, July 2002).

22. California Air Resources Board, *Hospitalizations and Emergency Room Visits Increase Following High Particulate Matter Episodes, Study Finds* (Sacramento: February 24, 2003), <http://www.arb.ca.gov/newsrel/nro22403.htm>.

23. For additional examples, see Schwartz, *Rethinking the California Air Resources Board's Ozone Standards*.

24. For data on asthma emergency room visits and hospitalizations by month, see, for example, Spokane Regional Health District, *Asthma in Spokane County* (Spokane, Washington: April 2002), <http://www.srhd.org/information/pubs/pdf/factsheets/AsthmaInSpokaneCounty.pdf>; J. Stockman, N. Shaikh, J. von Behren *et al.*, *California County Asthma Hospitalization Chart Book, Data from 1998-2000* (Sacramento: California Department of Health Services, September 2003), http://www.ehib.org/cma/papers/Hosp_Cht_Book_2003.pdf; Texas Department of Health, *Asthma Prevalence, Hospitalizations and Mortality — Texas, 1999-2001* (Austin: November 21, 2003), <http://www.tdh.state.tx.us/cphpr/asthma/asthma.pdf>; K. Tippy and N. Sonnenfeld, *Asthma Status Report, Maine 2002* (Augusta, ME: Maine Bureau of Health, November 25, 2002); K. R. Wilcox and J. Hogan, *An Analysis of Childhood Asthma Hospitalizations and Deaths in Michigan, 1989-1993* (Lansing, MI: Michigan Department of Community Health, undated), http://www.michigan.gov/documents/Childhood_Asthma_6549_7.pdf.

25. 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.

26. In 2004, EPA replaced the old 1-hour ozone standard with the significantly more stringent 8-hour standard.

27. The location is China Grove, in Rowan County.

28. In that year, the worst location was a rural site in Mecklenburg County. Rockwell, the second worst site in the state in 1998, had 27 exceedance days.
29. Carolinas Clean Air Coalition, *Impacts of Ozone on Our Health*.
30. The actual range in the study was 5 to 28 $\mu\text{g}/\text{m}^3$. However, $\text{PM}_{2.5}$ was measured using a different method from the one EPA began requiring in 1999 to determine compliance with the federal $\text{PM}_{2.5}$ standard. The CHS measured two-week-average $\text{PM}_{2.5}$ levels. This understates $\text{PM}_{2.5}$ levels, because it allows some “semi-volatile” species to evaporate, both because of the long collection time and because the filters are at ambient temperature. The new federal method measures daily average $\text{PM}_{2.5}$ and keeps the filters cooled to prevent evaporation. Because I compare the CHS $\text{PM}_{2.5}$ levels with $\text{PM}_{2.5}$ levels measured around the country using the new EPA method, I’ve corrected the CHS $\text{PM}_{2.5}$ measurements to make them equivalent to the EPA method. For details on the correction, see N. Motallebi, J. Taylor, B. E. Croes *et al.*, “Particulate Matter in California: Part 1 — Intercomparison of Several $\text{PM}_{2.5}$, $\text{PM}_{10-2.5}$, and PM_{10} Monitoring Networks,” *Journal of the Air & Waste Management Association* 53 (2003): 1509-16.
31. This percentage change is not explicitly mentioned in the journal article on the study. However, it can be calculated from information in the article. First, note from Table 3 that $\text{PM}_{2.5}$ 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 18 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 (see 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. Gauderman, Avol, Gilliland *et al.*, “The Effect of Air Pollution on Lung Development from 10 to 18 Years of Age.”
32. See Figure 3, *ibid.*
33. Once again, this is corrected for the low bias of dichotomous samplers relative to the newer FRM samplers. Motallebi, Taylor, Croes *et al.*, “Particulate Matter in California: Part 1 — Intercomparison of Several $\text{PM}_{2.5}$, $\text{PM}_{10-2.5}$, and PM_{10} Monitoring Networks.”
34. A. D. Rado, *Smog May Cause Lifelong Lung Deficits* (Los Angeles: University of Southern California, September 8, 2004), <http://www.usc.edu/uscnnews/stories/10495.html>.
35. Dr. Kenneth Olden, quoted in National Institutes of Health, “New Research Shows Air Pollution Can Reduce Children’s Lung Function,” September 9, 2004, <http://www.nih.gov/news/pr/sep2004/nihs-08a.htm>.
36. N. Bryant, “What Air Quality Problem?” *Charlotte Observer*, September 1, 2005, <http://www.charlotte.com/mld/charlotte/news/opinion/12530112.htm?BMIDS=13194>.
37. E-mail from Joel Schwartz to Nancy Bryant, September 7, 2004. On file with the author.
38. I. M. Goklany, *Clearing the Air: The Real Story of the War on Air Pollution* (Washington, DC: Cato, 1999).
39. Natural Resources Defense Council, *Breath-Taking: Premature Mortality Due to Particulate Air Pollution in 239 American Cities* (Washington, DC: May 1996), <http://www.nrdc.org/air/pollution/bt/btinx.asp>; R. Wilson and J. Spengler, *Particles in Our Air: Concentrations and Health Effects* (Cambridge, MA: Harvard University Press, 1996)..
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46. T. Lumley and L. Sheppard, "Time Series Analyses of Air Pollution and Health: Straining at Gnats and Swallowing Camels?" *Epidemiology* 14 (2003): 13-4.

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55. Newspapers carrying articles on the study included the *Los Angeles Times*, *Houston Chronicle*, *Philadelphia Inquirer*, and several others. The National Institutes of Health also put out a press release highlighting the study <http://www.nih.gov/news/pr/dec2005/niehs-22.htm>. For a more detailed critique, showing why the study is irrelevant for human or mouse PM_{2.5} risks and how the scientists involved misrepresented the study's results, see J. Schwartz, "Of Mice and Men," *Tech Central Station*, April 17, 2006, <http://www.tcsdaily.com/article.aspx?id=041706E>.

56. A few of the news stories mentioned that the study used "specially bred mice prone to heart disease." But this is a great understatement, because it creates the impression that the mice were similar to humans who have a high heart disease risk, and therefore that the study is relevant for human beings. In reality, the mice were genetically engineered to have cholesterol far beyond even the highest levels that would ever occur in humans or in "natural" mice.

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