POLICY REPORT

Where the Bodies are Buried

How experts for N.C.'s Attorney General mislead the public about TVA air pollution risks

Joel Schwartz

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Table of Contents

Cover photo is the Tennessee Valley Authority's Kingston Fossil Plant on the Tennessee River in Kingston, Tenn. Photo courtesy of the TVA.

Executive Summary

The Tennessee Valley Authority (TVA) operates 11 coal-fired power plants in the southeastern United States. These plants emit nitrogen oxides (NO_x) and sulfur dioxide (SO_2), which contribute to particulate matter (PM) and ozone in the eastern U.S., including North Carolina.

In an effort to force the TVA to reduce its contribution to air pollution in North Carolina, state Attorney General Roy Cooper filed suit against the TVA in January 2006, arguing that the TVA plants constitute a public nuisance.[1] To bolster his case, Cooper commissioned reports from experts in air pollution modeling (Lyle R. Chinkin and Neil J.M. Wheeler), control methods (James E. Staudt), health effects (John D. Spengler and Jonathan I. Levy), and cost-benefit analysis (Leland B. Deck).[2]

In reality, the actual benefits of the TVA power plant emission reductions will at best be only a tiny fraction of the amount claimed by the Attorney General's experts.

These experts estimated that reducing NO_x and SO_2 emissions from TVA sources by about 370,000 tons per year[3] (a 65 percent reduction) would have substantial health benefits, preventing more than 1,400 cases per year of premature mortality, more than a 1,000 hospital visits, and hundreds of thousands of asthma exacerbations each year.[4] By placing dollar values on these health benefits using standard cost-benefit analysis techniques, Leland Deck's expert report concluded that the health benefits would total $10.9 billion per year, or about 18 times greater than the annual cost of the emission reductions.[5]

In reality, the actual benefits of the TVA power plant emission reductions will at best be only a tiny fraction of the amount claimed by the Attorney General's experts. Nearly all the claimed health benefits of the emissions reductions come from reductions in fine particulate matter ($PM_{2.5}$). Particulate matter from power plants is mostly ammonium sulfate, formed from SO_2 emissions, with some ammonium nitrate, formed from NO_x emissions.[6] But neither of these substances is harmful, even at levels tens of times greater than are ever found in the air Americans breathe. In fact, ammonium sulfate is used as an "inert control"—that is, a substance that is not harmful—in studies of other substances that *are* expected to be toxic. According to the Attorney General's experts, 98.5 percent of the predicted health benefits of power plant emissions reductions are due to reductions in sulfate and nitrate particulates. In other words, 98.5 percent of the benefits claimed for power plant emissions reductions depend on the assumption that ammonium sulfate and nitrate are toxic. Since this assumption is false, 98.5 percent of the claimed benefits are not real.

The remaining 1.5 percent of predicted benefits comes from reductions in ozone pollution that are expected to result from reductions in NO_x emissions from the TVA power plants. Although ozone can be dangerous at high levels, the expert reports also exaggerate the benefits of ozone reductions. The key source of the exaggeration of benefits for ozone reductions is the experts' assumption that ozone causes premature death, even at the relatively low levels encountered in the air today. Based on decades of studies with several different species of animals, ozone is not deadly, even with long-term exposure to levels more than 13 times greater than the current federal ozone standard for daily peak levels. Reductions in premature mortality account for 96 percent of the total ozone benefits. Since ozone does not cause premature mortality, these benefits are likewise not real.

After calculating that the benefits of TVA emissions reductions would be 18 times the costs, Deck's expert report concludes "Such a large benefit/cost ratio is evidence that substantially different assumptions could be made in either the benefit or the cost analyses without changing my conclusion that the requested emission reductions do provide a substantial net benefit to society."[7]

This assertion is backwards. The high benefit/cost ratio is not evidence of net benefits. Rather, because sulfates and nitrates are harmless and because

ozone at today's low ambient levels is not deadly, the large benefit/cost ratio is evidence that false assumptions were used to generate it. Using the more realistic assumption that sulfates and nitrates are harmless eliminates 98.5 percent of the benefits. The more realistic assumption that low-level ozone is not deadly eliminates another 1.4 percent of the benefits. As a result, the costs of the TVA emission reductions will at best be more than 50 times greater than the benefits.

The Spengler and Levy expert report provided the air pollution health effects estimates that went into Deck's cost-benefit analysis. It is their selective omission and mischaracterization of evidence that ultimately resulted in the vast exaggeration of the health benefits of TVA power plant emissions reductions. The remainder of this report provides the detailed evidence refuting Spengler and Levy's incorrect claims about the health effects of TVA power plant emissions.

TVA Emissions Reductions

TVA emission reductions would reduce the sulfate and nitrate components of particulate matter, as well as ozone.

Particulate matter is made up of many substances, but the contribution from coal-fired power plants comes in the form of ammonium sulfate and ammonium nitrate. Through reactions in the atmosphere some of the SO_2 is converted to sulfate and some of the NO_x is converted to nitrate, both of which react with ammonia to form ammonium sulfate and nitrate. As the Spengler and Levy expert report notes sulfate is "the principal type of particulate air pollution produced from coal-fired power plant emissions." [8] NO_x also contributes to the formation of ozone.

The goal of the Attorney General's lawsuit is to force the TVA to reduce SO_2 and NO_x emissions from its power plants in order to reduce particulate matter and ozone. The expert report by Chinkin and Wheeler modeled the effect on $PM_{2.5}$ and ozone levels for the following TVA emission reduction scenario for the year 2013: SO_2 would be reduced from a baseline of 449,000 tons down to 137,000 tons, or a 69 percent reduction. NO_x would be reduced from a baseline of 115,000 tons down to 59,000 tons, or a 48 percent reduction. Spengler and Levy then predicted health improvements based on Chinkin and Wheeler's estimates of $PM_{2.5}$ and ozone reductions

Toxicity of Power Plant Emissions

Sulfate and nitrate particulate matter is harmless.

The claim that power plant emissions cause premature death, asthma attacks, hospital admissions, and other harms depends mainly on the toxicity of power plant particulates. Based on estimates in the expert reports, reductions in the components of $PM_{2.5}$ caused by TVA power plants accounts for 98.5 percent of all of the benefits attributed to emissions reductions from these plants, with the remaining 1.5 percent of benefits coming from reductions in ozone.[9]

However, as discussed in the previous section, the $PM_{2.5}$ caused by coal-fired power plants is in the form of ammonium sulfate, plus a small amount of ammonium nitrate. Neither of these substances is harmful. As a result, there are no health benefits from

reducing them. This eliminates 98.5 percent of the ostensible health benefits from the TVA emissions reductions.

The lack of toxicity of ammonium sulfate is well-known to air pollution health scientists. In fact, ammonium sulfate particulates are used as an "inert control"—that is, a substance that does not cause any harm and has no physiological effects—in studies of other potentially toxic agents. For example, Koenig et al. (1993) studied the effects of breathing sulfuric acid aerosols in elderly asthmatics and non-asthmatics.[10] As described in Koenig et al., "There were four test atmospheres: two included control exposures of air and *physiologically inert particles* (*ammonium sulfate*), and two included sulfuric acid…"[11] (emphasis added).

$PM_{2.5}$ caused by coal-fired power plants is in the form of ammonium sulfate, plus a small amount of ammonium nitrate. Neither of these substances is harmful. As a result, there are no health benefits from reducing them.

Indeed, the lack of toxicity of ammonium sulfate has been known for decades. During the late 1970s and early 1980s, researchers exposed human volunteers to as much as 1,000 micrograms per cubic meter ($\mu g/m^3$) of ammonium sulfate and yet found no physiological effects or harm.[12] For comparison, sulfate levels in U.S. air range from about 0.2 $\mu g/m^3$ to 7 $\mu g/m^3$ on an annual-average basis, and can get as high as about 30 $\mu g/m^3$ on the worst days at the worst locations in the U.S.[13] In other words, sulfate levels 30 times greater than the highest levels in U.S. air have no effects on human health. Indeed, asthma medications that open constricted airways are delivered in the form of inhaled sulfate aerosols—that is, in the form of sulfate particulate matter.[14]

Ammonium nitrate particulates have also proven to be non-toxic in human volunteers, even at levels tens of times greater than ever occur in the air people breathe.[15]

A number of recent scientific review articles have also noted the lack of toxicity of sulfate and nitrate particles in humans and laboratory animals, and the implausibility that these substances could be responsible for harmful effects.[16] Spengler and Levy omitted from their expert report the large body of evidence that sulfate and nitrate are harmless. As a result, they vastly exaggerate the benefits of TVA emissions reductions.

Leland Deck's expert report provides a cost-benefit analysis for the TVA emissions reductions and finds that the benefits are more than 18 times greater than the costs. Deck concludes "Such a large benefit/cost ratio is evidence that substantially different assumptions could be made in either the benefit or the cost analyses without changing my conclusion that the requested emission reductions do provide a substantial net benefit to society."[17]

This assertion is backwards. The high benefit/cost ratio is not evidence of net benefits. Rather, because sulfates and nitrates are harmless, the large benefit/cost ratio is evidence that false assumptions were used to generate it. Using the more realistic assumption that sulfates and nitrates are harmless eliminates 98.5 percent of the benefits and results in the costs of the emissions reductions being 3.6 times greater than the benefits. As detailed later in this report, Spengler and Levy also exaggerate harm from ozone. After removing Spengler and Levy's unrealistic assumptions, the estimated benefits of the TVA emissions reductions drop by more than 99.9 percent, making the costs more than 50 times greater than the benefits.

Attorney General's Experts Omit Contrary Evidence

Spengler and Levy mischaracterize study results and omit contrary evidence from their expert report.

Sulfate and nitrate are not the only cases where Spengler and Levy omit contrary evidence from their analysis. In addition, they mischaracterize many of the studies they cite in their expert report. Taken together, these tactics exaggerate the apparent harm from any given amount of air pollution and create a false appearance of consistency among studies in the research literature. Several examples follow:

Infant mortality studies. Spengler and Levy cite several studies that they claim support an overall conclusion that each 10 $\mu g/m^3$ increase in $PM_{2.5}$ is associated with a 7 percent increase in risk of premature death. One of these studies is Lipfert et al. (2000)[18], of which they state, "The study by Lipfert et al. reports numerous estimates but generally corroborates this magnitude of effect."

But here is what Lipfert et al. (2000) actually wrote: "Significant negative mortality associations were found for SO_4 [sulfate]." In epidemiological parlance, a "negative" association means that *higher* levels of the pollutant were associated with *lower* levels of harm. In other words, Lipfert et al. concluded that sulfate was not harmful—*exactly the opposite of how Spengler and Levy characterize Lipfert et al.*

Lipfert et. al. go on to write, "There was no indication of a role for outdoor $PM_{2.5}$, but possible contributions from indoor air pollution sources cannot be ruled out…." Thus, not only did Lipfert et al. not find any harm from power plant particulates specifically, they also found no evidence of harm from total $PM_{2.5}$.

Spengler and Levy rely mainly on Woodruff et al. (2006) and Ritz et al. (2006) for estimating infant mortality due to particulates, arguing that they are "the two strongest studies" of the relationship between air pollution levels and infant mortality.[9] However, both of these studies were of children in California, *where there are no coal-fired power plants and therefore no coal-related particulates.* Thus, even if sulfates or nitrates were toxic, these two studies would still be irrelevant for calculating the benefits of reducing emissions from the TVA's coal-fired power plants.

School absences. Spengler and Levy estimate that reducing ozone caused by NO_x emissions by TVA sources would prevent a total of 42,000 school absence days per year.[20] They based their estimate of the effect of ozone on school absences on Gilliland et al. (2001),[21] which reported results from the Children's Health Study (CHS). The CHS followed more than 2,000 children in 12 different California communities from 1993-2001.

What Spengler and Levy omit from their report is that two other studies failed to find an increase in school absences due to daily changes in ozone levels, even though these other two studies used the exact same CHS data as Gilliland et al., and even included some of the same authors.[22]

Spengler and Levy rely mainly on Woodruff et al. (2006) and Ritz et al. (2006) for estimating infant mortality .... However, both of these studies were of children in California, where there are no coal-fired power plants and therefore no coal-related particulates.

By citing one study that reported an association between ozone and absences and omitting the two studies that didn't, Spengler and Levy exaggerate the apparent harm from air pollution and create a false appearance of consistency in the research literature.

The fact that three studies using the exact same data came to wildly different conclusions is also an example of the degree to which the pollution associations reported in "observational" epidemiology studies are not representative of real causal effects, but are more a matter of subjective modeling choices by researchers and inherent limitations of observational methods. Spengler and Levy rely solely on observational epidemiology studies for their health-effects es-

timates, and this is one of the reasons why their claims are so wildly at odds with reality. The problems with observational studies will be discussed in more detail later in this report.

Beyond the problem of omitting contrary evidence on ozone and school absences, Spengler and Levy also ignore the fact that the Gilliland et al. (2001) ozone-absence relationship is not credible even on its own terms. For example, according to the Gilliland et al. results:

- Spending *more* time outdoors, which would have increased ozone exposures, was associated with *fewer* school absences.
- The apparent effects of ozone were associated mainly with ozone exposures from one or two *weeks* before the absence, rather than to ozone exposures within a few days before the absence.
- Higher levels of particulate matter were associated with a large increase in non-illness-related absences, but were not associated with an increase in absences due to illness. In fact, an increase of 10 $\mu g/m^3$ in PM_{10} (particulate matter under 10 microns in diameter) was associated with a larger effect on non-illness-related absences than the effect of a 0.020 ppm increase in ozone on respiratory-related absences.

Ozone levels peak during July and August .... Yet all across the U.S., July and August are also the months with the *lowest* rate of asthma ER visits.[26] In North Carolina, counties with higher ozone levels have lower rates of respiratory hospital admissions.

Taken together, these results are biologically implausible and suggest that the apparent effect of ozone on school absences was a statistical figment, rather than a real cause-and-effect relationship.

Asthma emergency room visits. Spengler and Levy predict that reductions in $PM_{2.5}$ from TVA power plants will prevent 250,000 asthma exacerbations per year.[23] Furthermore, they predict that reductions in $PM_{2.5}$ and ozone will prevent, respectively, 830 and 40 asthma emergency room (ER) visits.[24] As already noted, the $PM_{2.5}$ benefits are not real, because sulfate and nitrate particulates are not harmful.

The claimed benefits for ozone are also implausible. Ozone levels peak during July and August, because sunlight drives the reactions that form ozone.[25] Yet all across the U.S., July and August are also the months with the *lowest* rate of asthma ER visits.[26] In North Carolina, counties with higher ozone levels have lower rates of respiratory hospital admissions, as shown in **Figure 1**.

Spengler and Levy also mischaracterize the literature on air pollution and asthma attacks and omit contrary evidence. They cite five studies that they combined to generate their estimate that each 1 $\mu g/m^3$ increase in $PM_{2.5}$ is associated with a 2 percent increase in asthma exacerbations.[27] Spengler and Levy note that they gleaned these five studies from Table 8B-5 of the EPA's 2006 "Criteria Document" for ozone and conclude that "there is consistency in the magnitude of the concentration-response function across all studies."[28]

This claim might lead readers to believe that the five studies found similar and statistically significant associations between particulate matter and asthma exacerbations. But this is not the case. For example:

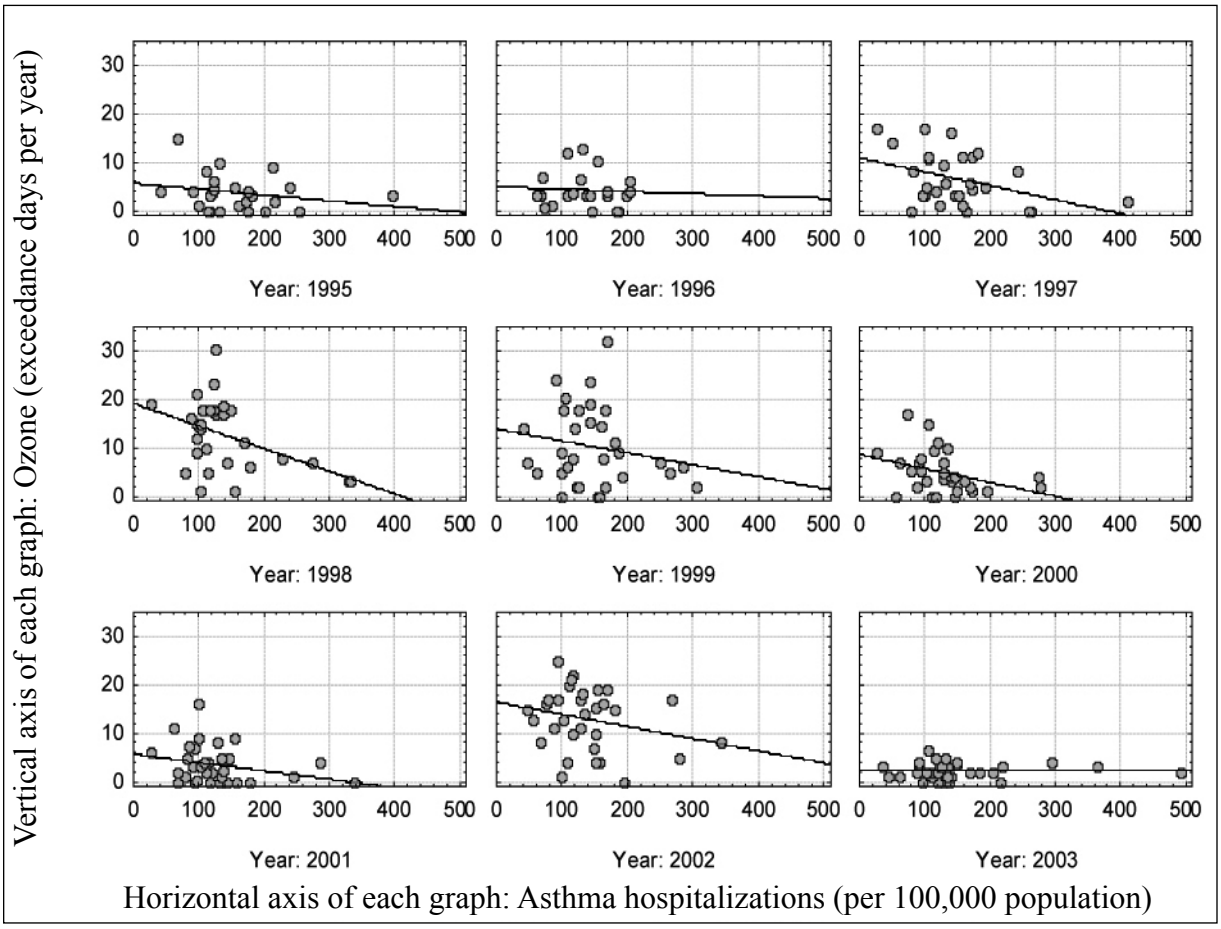
- In a study by Desqueyroux et al. (2002), increases in particulate matter during the previous two days were actually associated with a 33 to 50 percent *decrease* in asthma symptoms, though the effect was not statistically significant.[29] On the other hand, higher PM from four or five days ago was associated with an increase in asthma symptoms and this was statistically significant. This pattern is biologically implausible and suggests the result is a statistical figment, rather than a real effect.
- In Mortimer et al. (2002) the association be-

tween particulate matter and asthma symptoms was not even statistically significant.[30] Furthermore, the size of the putative PM effect decreased with time after exposure—just the opposite of the pattern reported by Desqueyroux et al.

Contrary to Spengler and Levy's claim, these studies are not consistent with each other, and they do not suggest a real effect of particulate matter on asthma symptoms.

Spengler and Levy also omit Gent et al. (2003), which found no relationship between $PM_{2.5}$ levels and asthma symptoms.[31] This is an interesting omission. Gent et al. was widely reported in the media when it was released because the study reported an association between higher ozone and increased asthma symptoms. Activists, regulators and many air pollution health scientists hailed the study as evidence that even low levels of ozone cause asthma exacerbations. But these same advocates omit Gent et al. when discussing the putative effects of $PM_{2.5}$ on asthma, because Gent et al. did not find any association between $PM_{2.5}$ and asthma symptoms. Likewise, when discussing ozone, advocates cite Gent et al. but omit studies, such as Mortimer et al. (2002), that did not find an effect of ozone on asthma symptoms. This selective citation of evidence creates an appearance of larger and more consistent harm from air pollution than warranted by

Figure 1: Asthma Hospitalization Rates vs. Ozone Level For N.C. Counties



Notes: Each point represents a given North Carolina county. Ozone exceedance days are based on an 8-hour, 0.085 ppm standard and are an average for all monitoring sites operating in a given county in a given year. Lines are linear regression lines.

Sources: Ozone data for North Carolina were downloaded from EPA's AIRData database, *http://www.epa.gov/air/data/geosel.html* (accessed September 29, 2006). Asthma hospitalization data were provided by the North Carolina State Center for Health Statistics.

the underlying weight of the research evidence.

Dublin coal-ban study. The city of Dublin, Ireland, in 1990 banned the use of soft (bituminous) coal for home heating and cooking, which resulted in a large drop in black smoke levels, particularly in winter. A study in the *Lancet* concluded that the coal ban caused a reduction in premature mortality.[32] Spengler and Levy cite this study to support their claim of health benefits from reducing particulates caused by coal-fired power plant emissions. They state "the reduction in respiratory and cardiovascular mortality that coincided with a ban on coal burning in Dublin, Ireland, is evidence that discernable effects of air pollution can be demonstrated on a population."[33]

Spengler and Levy are comparing apples and oranges. The coal in Dublin was burned inside homes for water and space heating, whereas coal in the U.S. is burned in large power plants. The difference is that the pollution issue in Dublin was the large amounts of black smoke (i.e., soot) produced by all of this domestic coal-burning, while sulfate and nitrate PM were not a factor. The situation is just the opposite in U.S. power plants, where the particulates from coal are non-toxic sulfate and nitrate formed in the atmosphere from the power plants' emissions of gaseous SO_2 and NO_x, rather than directly emitted black smoke and soot. Spengler and Levy's vague mention of "reduction in…mortality that coincided with a ban on coal" might mislead readers into thinking that pollution from domestic coal use in Dublin is comparable to power plant coal use in the U.S. when the two in fact bear no relationship to each other.

Observational Epidemiology Studies Versus Other Evidence

Observational epidemiology studies are invalid and at odds with more reliable evidence.

Given the direct evidence that sulfate and nitrate are not toxic, and that ozone is not deadly, one might ask how Spengler and Levy managed to create an appearance that their claims about harm from today's historically low levels of $PM_{2.5}$ and ozone are essentially a "done deal" with which no reasonable person could disagree.

Part of the answer has already been discussed. Spengler and Levy omit contrary evidence, claim some studies report harm from air pollution when in fact they don't, and ignore weaknesses in studies that do report harm from air pollution.

Nevertheless, it is still true that there are hundreds of studies in the scientific literature that report statistically significant associations between air pollution and health outcomes, such as risk of death, asthma attacks, etc. These studies are known as "observational" epidemiology studies—that is, studies that use data from non-randomly selected groups of people with non-random and poorly measured air pollution exposures and look for correlations between the two. All of Spengler and Levy's claims of health benefits from TVA emissions reductions are based on the results of observational studies.

The implicit assumption in an observational study is that after researchers have controlled for all known non-pollution factors that might be correlated with pollution levels and health outcomes (e.g., weather, smoking, diet, etc.) any remaining correlation between air pollution and health represents a genuine causal linkage between the two. A wide range of evidence shows that this assumption is false and that observational studies tend to "find" effects where no real effects exist.[34]

Indeed, many prominent epidemiologists are wringing their hands over the widespread problem and embarrassment of spurious health claims from observational epidemiology studies and are questioning whether observational studies are even capable of pro-

viding valid evidence on health risks.[35] Unfortunately, this acknowledgement of the limits of observational studies in the wider community of epidemiologists has had little effect on the relatively insular world of air pollution epidemiology specifically. Nevertheless, there have been some critiques even within air pollution epidemiology. Here for example is one caution on the validity of observational studies of air pollution's health effects:

> *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.***[36] (emphasis added)

The highlighted portion is key. Researchers make many subjective choices in developing statistical models relating air pollution to health. Furthermore, the studies are funded with the explicit goal of finding harm from air pollution. In this environment, researchers tend to choose statistical models that maximize the effect they "expect" or "hope" to find—a problem known as data-mining. As a result, observational studies become statistical fishing expeditions that turn up chance correlations rather than real effects.

An additional bias is that researchers are more likely to seek publication of, and journal editors are more likely to accept for publication, studies that find an effect, while studies that don't find any effects end up packed away into filing cabinets. The result is a problem known as "publication bias." The overall result is that the scientific literature includes lots of studies reporting "effects" that aren't real. Once again, some air pollution epidemiologists have noted the problem:

> *Publication bias arises because there are more rewards for publishing positive or at least statistically significant findings. It is a common if not universal problem in our research culture. In the case of time-series studies using routine data there are particular reasons why publication bias might occur. One is that the data are relatively cheap to obtain and analyse, so that there may be less determination to publish "uninteresting" findings. The other is that each study can generate a large number of results for various outcomes, pollutants and lags and there is quite possibly bias in the process of choosing amongst them for inclusion in a paper. In the field of air pollution epidemiology, the question of publication bias has only recently begun to be formally addressed.*[37]

In many areas of health research, randomized trials—a gold standard methodology that reduces or eliminates the biases inherent in observational studies—can be conducted to test claims made based on observational studies. In such cases, observational studies are routinely contradicted when checked with randomized trials.[38] In the case of air pollution, however, ethical and practical concerns make it impossible to do a randomized trial to test whether today's historically low air pollution levels are deadly. Still, if observational studies are invalid in all other areas of health research, there's no reason to expect them to do any better on air pollution.

A range of additional evidence also suggests that air pollution risks claimed in observational studies are spurious. Much of this evidence has already been presented, for example, the direct evidence that nitrate and sulfate particulate matter is harmless and the evidence that the health-effects literature is much less consistent and robust than Spengler and Levy claim.

Animal studies provide a further check. If air pollution at today's low ambient levels is deadly to people, then we would expect that much higher levels of air pollution would kill at least some laboratory

animals. However, researchers have not been able to kill various species of animals even with air pollution at levels many times greater than are ever found in ambient air. 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 US ambient levels."[39]

The same is true for ozone. The current federal ozone standard is 0.075 parts per (ppm), measured over an 8-hour average. But ozone does not kill animals, even after the equivalent of years of exposure to levels as high as 1.0 ppm—more than 13 times the federal ozone standard.[40]

A recent review of particulate matter toxicology concluded, "It remains the case that no form of ambient PM ... has been shown, experimentally or clinically, to cause disease or death at concentrations remotely close to US ambient levels."

Although observational air pollution studies in humans cannot be checked against randomized trials, a number of researchers have provided other types of direct evidence that observational studies are producing a false appearance of harm. For example, Spengler and Levy cite the American Cancer Society (ACS) study of $PM_{2.5}$ and mortality (Pope et al. (1995, 2002)) as evidence that any amount of particulate matter in the air is deadly.[41] The ACS study assessed the long-term effects of $PM_{2.5}$ exposure in different cities around the U.S. But reanalyses of the ACS data have demonstrated the extent to which observational studies can give spurious results when researchers leave out important confounding variables.

For example, in a reanalysis by the Health Effects Institute (HEI), 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.[42] Here's why: 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 original researchers. Regulators and air pollution epidemiologists (including the HEI researchers who did the reanalysis) have ignored this refutation of the ACS results and continue to claim the ACS study provides proof of harm from air pollution.

Spengler and Levy also cite the National Morbidity Mortality and Air Pollution Study (NMMAPS) to support the claim that daily fluctuations in ozone and PM are deadly.[43] NMMAPS assessed the association between daily ozone and PM levels and mortality in 95 U.S. cities. Spengler and Levy fail to mention that although NMMAPS reported a small association between higher air pollution and premature death on average, higher air pollution was associated with a *lower* risk of death in more than one-third of the 95 cities.[44]

One would never know about these problems with observational studies from reading Spengler and Levy's expert report. For example, they claim "The body of epidemiological literature for health effects of O_3 [ozone] and particulate matter is large and robust, and is supported by good mechanistic understanding of how these pollutants can influence human health. Premature mortality and morbidity attributable to fossil fuel-related particulate matter and O_3 persist in the U.S. and reduction in emissions from coal-fired power plants will have health benefits of the magnitude estimated within this report."[45] These claims are contradicted by large bodies of evidence summarized here, but omitted from Spengler and Levy's expert report.

Correcting the Experts' Errors and Exaggerations

Combining Spengler and Levy's health-benefit claims with Deck's cost-benefit analysis, reductions in sulfate and nitrate particulate matter account for 98.5 percent of the total benefits of TVA emissions reductions, while reductions in ozone account for the remaining 1.5 percent of benefits.[46] Of the ozone benefits, 1.4 percent accrue from reductions in premature mortality, and 0.1 percent from reductions in all of the other health effects attributed to ozone.

In this report, we have shown that sulfate and nitrate particulate matter are harmless, which means that 98.5 percent of the benefits claimed by the Attorney General's experts are not real. Accounting for the fact that ozone does not cause premature mortality eliminates another 1.4 percent of the benefits. We also showed that Spengler and Levy exaggerated other effects of ozone, such as school absences and emergency room visits, meaning that even the remaining 0.1 percent of benefits are exaggerated.

In sum, correcting the experts' false assumptions and mistaken health claims eliminates more than 99.9 percent of the benefits they claim for TVA power plant emissions reductions. As a result, the cost of those emissions reductions will be more than 50 times greater than the benefits.

Conclusion

North Carolina Attorney General Roy Cooper's experts claim that reducing emissions from the Tennessee Valley Authority's power plants will improve Americans' health to the tune of nearly $11 billion per year. But 98.5 percent of these benefits rely on the false assumption that sulfate and nitrate particulate matter is toxic. Another 1.4 percent of these benefits rely on the false assumption that ozone is deadly even at the relatively low levels found in U.S. air. By using these false assumptions and omitting contrary evidence, the Attorney General's experts made the benefits of power plant emissions reductions appear to be more than a thousand times greater than they actually are. After correcting the experts' false claims, the cost of the TVA emissions reductions is more than 50 times greater than the benefits.

Notes

1. North Carolina Department of Justice, "AG Cooper Seeks to Stop TVA from Unlawfully Polluting NC Air," press release, January 30, 2006, *http://www.ncdoj.com/DocumentStreamerClient?directory=PressReleases/file=clean%20air%20TVA%20suit%20final%20corrected%20version.pdf.* Also see, Elizabeth Shogren, "North Carolina Sues TVA to Clean Up Pollution," National Public Radio, November 1, 2006, *http://www.npr.org/templates/story/story.php?storyId=6417740.*

2. The four expert reports are: **John D. Spengler and Jonathan I. Levy,** *Public Health Benefits of Additional Emission Controls on Tennessee Valley Authority Coal-Fired Power Plants*, prepared for the North Carolina Department of Justice (Newton, Mass.: Environmental Health & Engineering, Inc., October 30, 2006); **Leland B. Deck**, *Economic Benefits Analysis of the Health Effects from Controlling Emissions from Tennessee Valley Authority Coal-Fired Power Plants*, prepared for the North Carolina Department of Justice (Washington, D.C.: Stratus Consulting, October 27, 2006); **James E. Staudt**, *Expert Report* [on cost of controlling emissions from TVA power plants], October 13, 2006; **Lyle R. Chinkin and Neil J. M. Wheeler**, *Air Quality Modeling and Analysis of Additional Emission Controls on Tennessee Valley Authority Coal-Fired Power Plants*, prepared for the North Carolina Department of Justice (Petaluma, CA: Sonoma Technology, August 29, 2006).

Each report addresses a different aspect of the issue, as follows:

Staudt: Cost of controlling NO_x and SO_2 emissions from TVA power plants.

Chinkin and Wheeler: Reduction in ambient levels of $PM_{2.5}$ and ozone due to the power plant emissions reductions.

Spengler and Levy: Reduction in premature mortality, hospital visits, and other health effects as a result of the power plant emissions reductions.

Deck: Dollar value of the health improvements and overall cost-benefit analysis of the power plant emission reductions.

For the sake of brevity, hereafter these reports will be cited, respectively as: Spengler and Levy; Deck; Staudt; and Chinkin and Wheeler.

3. The amount of emission reduction is based on the assumption that the TVA plants would have to meet the same requirements as power plants in North Carolina must meet under that state's Clean Smokestacks Act.

4. Spengler and Levy, Tables B.3 and B.4.

5. Deck and Staudt expert reports. These are the total benefits for all states affected by TVA power plant emissions. For North Carolina specifically, the estimated benefits amounted to $792 million. These are benefit estimates for the year 2013.

6. "Control of SO_2 contributes a significant majority of the mortality and morbidity benefits that we estimated for additional controls on the TVA plants." Spengler and Levy, p. 47.

7. Deck, p. 6.

8. Spengler and Levy, p. 13. In addition, Spengler and Levy later state "CMAQ model runs predicted lower concentrations of three components—sulfate, nitrate, and ammonium—of fine particles as a result of reduced emissions of SO_2 and NO_x from the coal-fired TVA facilities. Lower concentrations of sulfate, generally found in the form of ammonium sulfate, are attributable to reduced SO_2. Similarly, lower concentrations of nitrate, often in the form of ammonium nitrate, are attributable to reduced emissions of NO_x."

9. The remaining benefits come from ozone reductions, which are discussed later in this report. The portion of benefits attributed respectively to $PM_{2.5}$ and ozone were calculated by multiplying the number of cases of each health effect prevented (Spengler and Levy, Tables B.3 and B.4) by the dollar cost attributed to each health effect (Deck, Table 6, using the column for 2006 prices and incomes).

10. J. Q. Koenig, K. Dumler, V. Rebolledo et al., "Respiratory Effects of Inhaled Sulfuric Acid on Senior Asthmatics and Nonasthmatics," *Archives of Environmental Health* 48 (1993): 171-5.

11. The parenthetical "(ammonium sulfate)" is in the original publication. Ibid.

12. For example, Sackner et al. (1977) exposed volunteers to 1,000 µg/m^3 (sic) of sulfate particulate matter and concluded "Brief exposure to microaerosols of sulfate particulate matter do not appear to adversely affect cardiopulmonary function of normal humans even in concentrations up to 20 times greater than the highest environmental urban concentrations recorded." Utell et al. (1983) had 17 asthmatics inhale ammonium sulfate and other sulfates at levels ranging from 100 µg/m^3 to 1,000 µg/m^3 and concluded: "At the 1,000 µg/m^3 concentration, the Threshold Limit Value for occupational exposure, H_2SO_4 [sulfuric acid] and NH_4HSO_4 [ammonium bisulfite] inhalation produced significant reductions in specific airway conductance (SGaw) (p less than 0.05) and forced expiratory volume in one second (p less than 0.01) compared with NaCl [table salt] or pre-exposure values. At the 450 µg/m^3 concentration, only H_2SO_4 inhalation produced a significant reduction in SGaw (p less than 0.01). At 100 µg/m^3, a level 3 to 5 times greater than peak urban levels, no significant change in airway function occurred after any sulfate exposure." M. A. Sackner, D. Ford and R. Fernandez, "Effect of Sulfate Aerosols on Cardiopulmonary Function of Normal Humans," *American Review of Respiratory Diseases* 115 (1977): 240; M. J. Utell, P. E. Morrow, D. M. Speers et al., "Airway Responses to Sulfate and Sulfuric Acid Aerosols in Asthmatics. An Exposure-Response Relationship," *American Review of Respiratory Disease* 128 (1983): 444-50.

13. W. H. White, L. L. Ashbaugh, N. P. Hyslop et al., "Estimating Measurement Uncertainty in an Ambient Sulfate Trend," *Atmospheric Environment* 39 (2005): 6857-67.

14. See, for example, M. Blitz, S. Blitz, R. Hughes et al., "Aerosolized Magnesium Sulfate for Acute Asthma: A Systematic Review," *Chest* 128 (2005): 337-44.

15. M. T. Kleinman, W. S. Linn, R. M. Bailey et al., "Effect of Ammonium Nitrate Aerosol on Human Respiratory Function and Symptoms," *Environmental Research* 21 (1980): 317-26; R. W. Stacy, E. Seal, Jr., D. E. House et al., "A Survey of Effects of Gaseous and Aerosol Pollutants on Pulmonary Function of Normal Males," *Archives of Environmental Health* 38 (1983): 104-15; M. J. Utell, A. J. Swinburne, R. W. Hyde et al., "Airway Reactivity to Nitrates in Normal and Mild Asthmatic Subjects," *Journal of Applied Physiology* 46 (1979): 189-96.

16. L. C. Green and S. R. Armstrong, "Particulate Matter in Ambient Air and Mortality: Toxicologic Perspectives," *Regulatory Toxicology and Pharmacology* 38 (2003): 326-35; R. B. Schlesinger, "The Health Impact of Common Inorganic Components of Fine Particulate Matter ($PM_{2.5}$) in Ambient Air: A Critical Review," *Inhalation Toxicology* 19 (2007): 811-32; R. B. Schlesinger and F. Cassee, "Atmospheric Secondary Inorganic Particulate Matter: The Toxicological Perspective as a Basis for Health Effects Risk Assessment," *Inhalation Toxicology* 15 (2003): 197-235.

17. Deck, p. 6.

18. F. W. Lipfert, J. Zhang, and R. E. Wyzga, "Infant Mortality and Air Pollution: A Comprehensive Analysis of U.S. Data for 1990," *Journal of the Air and Waste Management Association* 50 (2000): 1350-66.

19. Spengler and Levy, p. 35. B. Ritz, M. Wilhelm, and Y. Zhao, "Air Pollution and Infant Death in Southern California, 1989-2000," *Pediatrics* 118 (2006): 493-502; T. J. Woodruff, J. D. Parker, and K. C. Schoendorf, "Fine Particulate Matter ($PM_{2.5}$) Air Pollution and Selected Causes of Postneonatal Infant Mortality in California," *Environmental Health Per-*

spectives 114 (2006): 786-90.

20. Spengler and Levy, Table B.4.

21. Spengler and Levy, p. 30. F. D. Gilliland, K. Berhane, E. B. Rappaport et al., "The Effects of Ambient Air Pollution on School Absenteeism Due to Respiratory Illnesses," *Epidemiology* 12 (2001): 43-54.

22. The two studies are K. Berhane and D. C. Thomas, "A Two-Stage Model for Multiple Time Series Data of Counts," *Biostatistics* 3 (2002): 21-32; V. Rondeau, K. Berhane, and D. C. Thomas, "A Three-Level Model for Binary Time-Series Data: The Effects of Air Pollution on School Absences in the Southern California Children's Health Study," *Statistics in Medicine* 24 (2005): 1103-15.

23. An "exacerbation" means increases in symptoms such as coughing, wheezing, and chest tightness. Spengler and Levy, Table B. 3.

24. Spengler and Levy, Tables B. 3 and B.4.

25. In the eastern half of the U.S., summer is also the season with the highest particulate levels.

26. 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* (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://archive.dshs.state.tx.us/legacytdh/cphpr/asthma.htm*; K. Tippy and N. Sonnenfeld, *Asthma Status Report, Maine 2002* (Augusta, Maine: 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, Mich.: Michigan Department of Community Health, undated), *http://www.michigan.gov/documents/Childhood_Asthma_6549_7.pdf*.

27. Spengler and Levy, p. 38.

28. The Criteria Document is EPA's review of ozone health effects science to support tightening the ozone standard. Environmental Protection Agency, *Air Quality Criteria for Ozone and Related Photochemical Oxidants* (Washington, DC: February 2006), *http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=149923*.

29. H. Desqueyroux, J. C. Pujet, M. Prosper et al., "Short-Term Effects of Low-Level Air Pollution on Respiratory Health of Adults Suffering from Moderate to Severe Asthma," *Environmental Research* 89 (2002): 29-37.

30. K. M. Mortimer, L. M. Neas, D. W. Dockery et al., "The Effect of Air Pollution on Inner-City Children with Asthma," *European Respiratory Journal* 19 (2002): 699-705.

31. Spengler and Levy only included studies that reported an overall daily "symptom score" for asthma. Gent et al. (2003) reported results for each symptom separately (e.g., chest tightness, wheezing, etc.). J. F. Gent, E. W. Triche, T. R. Holford et al., "Association of Low-Level Ozone and Fine Particles with Respiratory Symptoms in Children with Asthma," *Journal of the American Medical Association* 290 (2003): 1859-67.

32. L. Clancy, P. Goodman, H. Sinclair et al., "Effect of Air-Pollution Control on Death Rates in Dublin, Ireland: An Intervention Study," *Lancet* 360 (2002): 1210-4.

33. Spengler and Levy, p. 13.

34. S. Begley, "New Journals Bet 'Negative Results' Save Time, Money," *Wall Street Journal*, September 15, 2006, B1, *http://online.wsj.com/article/SB115827169620563571-email.html*; J. P. Ioannidis, "Why Most Published Research Findings Are False," *PLoS Med* 2 (2005): e124; J. P. A. Ioannidis, "Contradicted and Initially Stronger Effects in Highly Cited Clinical Research," *Journal of the American*

Medical Association 294 (2005): 218-28; G. Taubes, "Epidemiology Faces Its Limits," *Science* 269 (1995): 164-69; G. Taubes, "Do We Really Know What Makes Us Healthy?" *New York Times*, September 16, 2007, *http://www.nytimes.com/2007/09/16/magazine/16epidemiology-t.html?_r=3&ref=magazine&oref=slogin&oref=slogin&oref=slogin*.

34. S. Ebrahim and M. Clarke, "STROBE: New Standards for Reporting Observational Epidemiology, a Chance to Improve," *International Journal of Epidemiology* 36 (2007): 946-48; S. J. Pocock, T. J. Collier, K. J. Dandreo et al., "Issues in the Reporting of Epidemiological Studies: A Survey of Recent Practice," *British Medical Journal* 329 (2004): 883; G. D. Smith and S. Ebrahim, "Epidemiology - Is It Time to Call It a Day?" *International Journal of Epidemiology* 30 (2001): 1-11; E. von Elm and M. Egger, "The Scandal of Poor Epidemiological Research," *British Medical Journal* 329 (2004): 868-69.

36. T. Lumley and L. Sheppard, "Time Series Analyses of Air Pollution and Health: Straining at Gnats and Swallowing Camels?" *Epidemiology* 14 (2003): 13-4.

37. H. Anderson, R. Atkinson, J. Peacock et al., *Meta-Analysis of Time-Series Studies and Panel Studies of Particulate Matter* (PM) *and Ozone* (World Health Organization, 2004), *www.euro.who.int/document/e82792.pdf*.

38. Begley, "New Journals Bet 'Negative Results' Save Time, Money."; Ioannidis, "Why Most Published Research Findings Are False."; Taubes, "Do We Really Know What Makes Us Healthy?"

39. Green and Armstrong, "Particulate Matter in Ambient Air and Mortality: Toxicologic Perspectives."

40. During EPA's review of the federal ozone standard, UC Davis professor Charles Plopper, a member of EPA's Clean Air Science Advisory Committee, noted that for decades he had been exposing animals to very high concentrations of ozone but that he had never killed any. As Professor Plopper said, "I'm trying to look at it as a biologist and trying to figure out whether [ozone] exposure kills people. And I've never killed a rat in 35 years…never killed a monkey in 35 years" (transcript of CASAC meeting, December 8, 2005, p. 148). Indeed, dozens of studies have been performed on monkeys, rats, mice, dogs, and other animals, with daily exposures as high as 1.0 ppm and often continuing for the equivalent of years, but none has reported any deaths, despite the enormous ozone exposures.

41. Spengler and Levy, p. 16. C. A. Pope, M. J. Thun, M. M. Namboodiri et al., "Particulate Air Pollution as a Predictor of Mortality in a Prospective Study of U.S. Adults," *American Journal of Respiratory and Critical Care Medicine* 151 (1995): 669-74; C. A. Pope, R. T. Burnett, M. J. Thun et al., "Lung Cancer, Cardiopulmonary Mortality, and Long-Term Exposure to Fine Particulate Air Pollution," *Journal of the American Medical Association* 287 (2002): 1132-41.

42. D. Krewski, R. T. Burnett, M. S. Goldberg et al., *Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality* (Cambridge, Mass.: Health Effects Institute, July, 2000).

43. Spengler and Levy, p. 28.

44. Of particular interest for North Carolina, higher ozone was associated with slightly lower risk of death in Raleigh (not statistically significant) and no change in risk in Charlotte. Higher PM was associated with a higher risk of death in Charlotte and a lower risk of death in Raleigh (neither statistically significant). F. Dominici, A. McDermott, M. Daniels et al., *Revised Analyses of the National Morbidity, Mortality, and Air Pollution Study, Part II* (Boston: Health Effects Institute, May 2003); Environmental Protection Agency, *Air Quality Criteria for Ozone and Related Photochemical Oxidants*.

45. Spengler and Levy, p. 49.

46. Calculated from Deck, Table 6 and Spengler and Levy, Tables B.3 and B.4. See note 9 for details.

About the Author

Joel Schwartz is a visiting scholar at the American Enterprise Institute and the author of "Air Quality in America: A Dose of Reality on Air Pollution Levels, Trends, and Health Risks" (AEI Press, 2008).

Mr. Schwartz formerly directed the Reason Public Policy Institute's Air Quality Project and has also published studies on chemical risks and extended producer responsibility. Prior to joining Reason, he was Executive Officer of the California Inspection and Maintenance Review Committee, a government agency charged with evaluating California's vehicle emissions inspection program and making recommendations to the legislature and governor on program improvements. He has worked at the RAND Corporation, the South Coast Air Quality Management District, and the Coalition for Clean Air.

Mr. Schwartz received his bachelor's degree in chemistry from Cornell University and his master's degree in planetary science from the California Institute of Technology.

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"To prejudge other men's notions before we have looked into them is not to show their darkness but to put out our own eyes."

JOHN LOCKE (1632-1704)

Author, Two Treatises of Government and Fundamental Constitutions of Carolina

John Locke Foundation

200 West Morgan St.
Raleigh, NC 27601
V: 919-828-3876
F: 919-821-5117
www.johnlocke.org
info@johnlocke.org