Burying Evidence

The Union of Concerned Scientists' Unscientific Claims about Air Pollution and Health

Joel Schwartz

Visiting Fellow

American Enterprise Institute

January 16, 2007

Introduction

In *Digging Up Trouble: The Health Risks of Construction Pollution in California*, the Union of Concerned Scientists (UCS) claims air pollution from construction vehicles is killing more than 1,100 Californians each year, sending similar numbers to the hospital, and sickening hundreds of thousands more.[1] UCS estimates the economic toll at more than $9 billion per year. Fortunately, these claims have little to do with reality. UCS exaggerates harm from air pollution by excluding contrary evidence and ignoring weaknesses in studies that support its predetermined conclusions.

According to UCS, the harm from construction emissions results mainly from two air pollutants: particulate matter (PM) and ozone. PM can be directly emitted (e.g., diesel smoke) or formed in the atmosphere from gaseous emissions (e.g., nitrogen oxides (NOx) can be converted to particulate nitrate). The California Air Resources Board (CARB) estimates that construction equipment contributes 3 percent of statewide direct fine particulate matter ($PM_{2.5}$) emissions and 28 percent of $PM_{2.5}$ emissions from diesel vehicles specifically.[2] Ozone is not directly emitted, but is formed in the atmosphere through reactions of NOx and volatile organic compounds (VOC) in the presence of sunlight. CARB estimates that construction equipment contributed 11 percent of statewide NOx emissions and 5 percent of VOC in 2005.[3]

Construction equipment is thus a significant contributor to total air pollutant emissions. Nevertheless, the actual harm from these emissions is far lower than UCS claims:

- Laboratory studies indicate that current, historically low levels of air pollution are at worst a minor factor in people's health.[4] Health researchers have been unable to kill laboratory animals even with particulate matter at concentrations many times greater than the most polluted California air. Laboratory studies with human volunteers, including asthmatics, have not found harm from $PM_{2.5}$ even at concentrations a few times greater than the highest real-world levels. This is true even for components of PM, such as diesel soot, that would be expected to have the highest toxicity. UCS does not mention or include any of this evidence in its report.

- Instead, UCS bases its health claims on the results from a much weaker type of study design called an "observational" epidemiology study. Observational studies work with non-randomly selected subjects and non-randomly assigned pollution exposures and then use statistical techniques to try to remove the biases inherent in non-random data. Unfortunately, a range of evidence shows that observational studies are unreliable and tend to create an appearance of risk where no risk in fact exists. UCS does not mention the weaknesses in its chosen form of evidence. Furthermore, even with their inherent biases, many observational studies have not found any harm associated with air pollution, yet UCS omits this contrary evidence from its analysis as well.

- UCS assumes that NOx emissions from construction equipment increase ozone, but in fact NOx emissions *reduce* ozone. A range of air pollution research has

shown that when the ratio of VOC to NOx in air is relatively low—a condition typical in California's metropolitan areas—reducing NOx *increases* ozone, and vice versa. The key evidence is that total NOx levels decline substantially on weekends, mainly due to reductions in the use of diesel trucks and construction equipment, but ozone levels rise.

- UCS exaggerates Californians' exposure to air pollution. For example, UCS claims "more than 90 percent of Californians live in areas that do not comply with the federal ozone standard." The real percentage is only one-third of what UCS claims. UCS generated its exaggerated value by counting "clean" areas as "dirty." For example, even though 99 percent of people in San Diego County live in areas that comply with the federal 8-hour ozone standard, UCS counts all 3 million San Diegans as living in an area that violates the standard. Thus, in addition to exaggerating the harm from any given level of air pollution, UCS also exaggerates the air pollution levels themselves.

At high enough concentrations diesel exhaust can be an unpleasant and aggravating nuisance. But this is a far cry from UCS's accusation that more than a thousand people are killed each year or that hundreds of thousands suffer serious harm from construction-related emissions.

UCS has vilified the Bush administration, sometimes with good reason, for manipulating scientific research for political purposes, and has even created a whole campaign and Web site to expose and condemn the politicization of science. Yet, in *Digging Up Trouble* UCS itself puts on a clinic in the selective use of scientific evidence to reach predetermined conclusions and support extra-scientific political goals.[5] The remainder of this commentary provides a more detailed critique of UCS's misleading account of the health effects of current, historically low air pollution levels.[6]

Evaluating the Real Risks

UCS attributes 98 percent of the harm from construction emissions to premature deaths supposedly caused by $PM_{2.5}$ and ozone.[7] But these deaths are statistical figments rather than real harm from air pollution.

UCS implicitly attributes about 40 percent of the air pollution-related deaths from construction equipment to nitrate PM caused by NOx emissions.[8] However, laboratory research on animals and human volunteers indicates that nitrates are not toxic, even at levels many times greater than ever occur in the most polluted California air.[9] UCS assumes all particulate matter has the same health effects, regardless of composition, and does not mention any of the evidence showing that nitrate PM is not harmful. Right off the bat these data reduce UCS's death claim by 40 percent.

UCS attributes another 10 percent of deaths to ozone caused by NOx and VOC emissions.[10] But emissions from construction equipment actually cause a net *decrease* in ozone. The reason is that when there is a low ratio of VOC to NOx in air, NOx becomes a net ozone destroyer. Under this circumstance, *reducing* NOx actually

increases ozone.[11] This is the situation in much of California and has been for at least a decade. For example, in the Los Angeles region, NOx levels are about 25 percent to 40 percent lower on Sundays than on weekdays, but ozone levels are 20 percent to 50 percent higher.[12] Even though weekends account for only 29 percent of all days of the year, nearly 50 percent of 8-hour ozone exceedance days in the Los Angeles metro area occur on weekends. San Diego and the San Francisco Bay Area similarly have lower NOx and higher ozone on weekends.

NOx levels drop so much on weekends because diesel vehicles—such as construction equipment—are a large source of NOx and these vehicles are much less active on weekends.[13] The evidence suggests that NOx reductions are the cause of the increase in weekend ozone levels.[14] Thus, regardless of the health effects of ozone, *construction emissions reduce ozone*. Knock off another 10 percent of the deaths and health costs UCS claims for construction emissions. Despite its claim to be a group of scientists that bases its claims on scientific research, UCS does not mention any of the substantial scientific literature on the role of NOx emissions in *reducing* ozone levels in California.

Diesel smoke is more noxious than nitrate PM, as anyone who has ever stood near the exhaust pipe of an old school or transit bus can attest. Yet, even diesel smoke and $PM_{2.5}$ in general show little evidence for harm at the relatively low exposure levels that occur in the real world today. For example, two separate Health Effects Institute (HEI) studies exposed both healthy and asthmatic human volunteers to 100 $\mu g/m^3$ of diesel particulate matter (DPM) and 200 $\mu g/m^3$ of Los Angeles-area $PM_{2.5}$ for 2 hours while they exercised.[15]

Both of these are high exposures when compared with $PM_{2.5}$ levels people out in the real world experience. Recent measurements next to one of the busiest freeways in Los Angeles found that black carbon, a major component of diesel smoke, never exceeded 10 $\mu g/m^3$ and averaged 5.4 $\mu g/m^3$.[16] In terms of total $PM_{2.5}$, even Riverside, California, which has the highest $PM_{2.5}$ levels in the United States, never reaches 200 $\mu g/m^3$ of total $PM_{2.5}$ and only rarely exceeds even 100 $\mu g/m^3$. Despite the relatively high particulate exposure levels in the HEI study, the researchers did not find changes in symptoms or lung function in either the healthy or asthmatic subjects.

Animal studies can use much higher PM levels than studies with human volunteers. Yet diesel soot and ambient $PM_{2.5}$ do not cause premature death in animals until concentrations reach levels tens to hundreds of times greater than would ever be experienced in ambient air.[17] As a recent review 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.*[18]

Thus, the weight of the evidence from controlled studies with animals and human volunteers suggests that PM is unlikely to cause premature death or other serious health effects at levels found in real-world air. UCS does not mention any of these research

results or even imply that there is any evidence at all against the claims it makes in *Digging Up Trouble*.

The studies discussed above randomly assigned subjects to "treatment" and "control" groups. Random assignment ensures that the treatment and control groups differ only in whether they are exposed to air pollution. Thus, any observed health effects can be more confidently attributed to air pollution and not to other unrelated factors. This type of study is the "gold standard" for sorting out whether a given factor—for example, a new drug, a change of diet, an air pollutant, etc.—really affects health.

Like UCS, other environmental activists, as well as government regulators, have ignored the evidence from controlled studies. Instead, they cite results from a much weaker type of study design called an "observational" study. Observational studies work with non-randomly selected subjects and non-randomly assigned pollution exposures and then use statistical methods to try to remove the biases inherent in non-random data. Most epidemiological studies you read about in the newspaper—studies that assess the effects of diet or health habits on risk of cancer or heart disease, for example—are of this non-randomized, observational sort.

The output of an observational epidemiology study is a correlation between some factor, say air pollution levels or dietary fat, and a health outcome, such as death, atherosclerosis, or an asthma attack. But unlike controlled laboratory studies, which produce direct evidence for cause-effect relationships, the evidence from observational studies is indirect. The implicit assumption in an observational study is that after researchers have controlled for all known sources of bias, any residual correlation between, say, air pollution and risk of death represents a genuine causal connection. However, several lines of evidence indicate that this assumption is false, and that observational studies instead tend to turn up false indications of risk.

Publication Bias and Data Mining

First, it is nearly impossible to control for all of the biases inherent in non-random data, because most of these biases are either unmeasured or unknown. Second, phenomena known as "publication bias" and "data mining" exaggerate the apparent size of any given health effect reported in the epidemiologic literature and encourage researchers to "find" what they are looking for.

Publication bias refers to the tendency of researchers to seek publication of, and for scientific journals to accept for publication, mainly those studies that find a statistically significant effect, while not publishing studies that do not find an effect. As a result, the real effect of any particular air pollutant, diet, medical intervention, etc., is smaller than the studies in the scientific literature would naïvely lead one to believe.

Data mining refers to the risk that observational studies can become statistical fishing expeditions that turn up chance correlations, rather than real causal relationships. Think of the statistical models that researchers use to control for bias in observational studies as

having lots of "dials" or "knobs" that the researchers can turn in order to "tune" the statistical model until it fits the observations. Within the presumed uncertainties in the data and methods, researchers tend to turn these knobs and dials in ways that maximize the effects they "expect" or "hope" to find, and are more likely to seek publication of studies that find the expected effect.

Researchers have been aware of these problems for a long time.[19] Here is a recent caution on publication bias from a group of air pollution epidemiologists:

> *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 field of air pollution epidemiology, the question of publication bias has only recently begun to be formally addressed.*[20]

Air pollution epidemiologists have also noted that it is common for researchers to selectively report results for statistical models that maximize the apparent risks of air pollution, rather than the full ensemble of results of their statistical modeling:

> *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.*[21] (emphasis added)

> *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.*[22]

Publication bias and data mining are not merely speculative concerns. They are serious problems in air pollution epidemiology and health research in general. In just the last few years much conventional medical wisdom that was based on observational epidemiology studies has been tested and overturned by randomized controlled trials that eliminate the biases inherent in observational studies.[23] Spurious results from observational studies have become such a pervasive problem in the medical literature that health researchers have been creating new journals specifically designed to combat publication bias and data mining.[24] A number of epidemiologists believe that observational epidemiology methods are not even capable of providing reliable evaluations of health risks, especially when the putative risks are relatively small, as they are for air pollution.[25]

Epidemiologists have also provided direct evidence that observational studies of air pollution and health are generating false indications of risk.[26] Furthermore, the key observational studies that regulators and activists use to justify their air pollution health claims suffer from spurious and biologically implausible results.

For example, UCS cites two research reports from the American Cancer Society (ACS) study of particulate matter and mortality as the evidence for premature death from long-term exposure to $PM_{2.5}$.[27] But these same two reports concluded that $PM_{2.5}$ appeared to kill men but not women, those who said they were moderately active but not those who said they were either very active or sedentary, and those with no more than a high school degree but not those with at least some college-level education. These biologically implausible outcomes suggest that the ACS results reflect uncontrolled statistical biases rather than real harm from pollution.

The Health Effects Institute (HEI) performed sensitivity analyses on the ACS data that provided additional evidence that its results were merely statistical artifacts. For example, when migration rates into and out of various cities over time were added to the ACS statistical model relating $PM_{2.5}$ and risk of death, the apparent effect of $PM_{2.5}$ disappeared.[28] 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 relatively healthier people leaving cities with higher-than-average pollution levels. Migration was just one of several confounding factors that diminished or erased the apparent harm from $PM_{2.5}$ but were not accounted for by the ACS researchers. Incidentally, UCS ignores two other major studies that did not find any harm from long-term $PM_{2.5}$ exposure.[29]

Another HEI effort, the National Morbidity, Mortality and Air Pollution Study (NMMAPS), reported that in about one-third of the 90 cities evaluated, higher levels of particulate matter and ozone were associated with lower risks of premature death.[30] How could air pollution kill people in some cities but save them in others? More likely both effects are the spurious result of uncontrolled statistical biases.

Not the Whole Truth

Digging Up Trouble includes many more examples of UCS exaggerating or cherry-picking the evidence. For example, UCS claims "as much as 10 to 20 percent of all summertime hospital visits and admissions for respiratory illness are associated with ozone…"[31] But not even CARB or EPA claim anywhere near this large a health burden from ozone and UCS claims to base its health effects estimates on the same studies that CARB and EPA use.[32] When CARB adopted a tougher ozone standard for California, agency staff estimated that eliminating virtually all human-caused ozone in the state would reduce asthma-related emergency-room visits by 1.75 percent and respiratory hospital admissions by 1.2 percent.[33] EPA scientists estimated similarly small health benefits from reducing ozone.[34] Compared to the regulators' estimates, UCS overstates the harm from ozone by at least a factor of six.

But even the small impact of ozone claimed by CARB and EPA is still an exaggeration of the real harm, because both agencies ignored contrary evidence. For example, when assessing the potential benefits of a tougher ozone standard, CARB’s staff omitted a

study in California's Central Valley that found that higher ozone was associated with a *lower* rate of hospital visits.[35] CARB was certainly aware of the existence of this study, because CARB funded and published it.

According to *Digging Up Trouble* $PM_{2.5}$ also contributes to respiratory hospital visits and asthma symptoms.[36] But UCS ignores a study of several hundred asthmatic children in Connecticut that did not find any association between $PM_{2.5}$ and asthma symptoms.[37]

The two studies just cited, the Central Valley study and the Connecticut study, are signal examples of how the overall evidence in the research literature is far more equivocal than advocates make it appear. The Central Valley study reported harm from PM, but not ozone. The Connecticut study reported harm from ozone, but not PM. Regulators and activists mention only the PM results from the Central Valley study and only the ozone results from the Connecticut study, creating an appearance of consistency and robustness in the research base that does not in fact exist.

Data from California and elsewhere in the United States also show that hospital visits for asthma attacks are *lowest* in July and August—the months when ozone concentrations are at their *highest*.[38] UCS ignores this evidence as well.

UCS claims that ozone from construction emissions causes more than 300,000 school absence days each year.[39] As shown above, construction emissions actually *reduce* ozone. Regardless, UCS was selective in choosing its evidence on whether higher ozone is associated with an increase in school absences. UCS cites a CARB health effects report as the source its claims of school absences due to ozone.[40] CARB in turn cites Gilliland et al. (2001), which used data from CARB's Children's Health Study (CHS), a long-term study of thousands of California children living in communities with a wide range of pollution levels.[41]

CARB and UCS ignored the biological implausibility of the results in Gilliland et al. For example, an absence from school on a given day appeared to be due mainly to ozone levels from one or two weeks ago, rather than ozone levels during the previous few days. Spending more time outdoors, which would have increased ozone exposures, was paradoxically associated with *fewer* school absences. Particulate matter was associated with a large increase in *non-illness-related* absences, but not with absences due to illness. Taken as a whole, the study's results are not credible and are an additional example of the problems with observational studies.

UCS and CARB also fail to mention that two other studies have been published using the exact same CHS dataset, but did not find an association between ozone and school absences.[42] This is another example not only of UCS's selective use of evidence to support its pre-determined conclusions, but also of the unreliability of observational studies for assessing health risks, since three different studies using the same data came up with three different results.[43]

Overexposure

In addition to exaggerating the health effects of any given level of air pollution, UCS creates a false appearance that elevated air pollution is more widespread than it really is. According to UCS "more than 90 percent of Californians live in areas that do not comply with the federal ozone standard." This is one of those claims that contains a technical grain of truth, but that leads readers to draw conclusions that are false.

EPA and CARB classify entire regions as "non-attainment" areas under the Clean Air Act even if only a single pollution monitor in the region violates a federal pollution standard. This makes sense from a regulatory perspective, because emissions in one part of a region can affect pollution levels in other parts. But UCS's implication here is that more than 90 percent of Californians actually breathe air that does not comply with the federal ozone standard. This claim is high by about a factor of three.

For example, San Diego County violates the federal 8-hour ozone standard, but only at a single rural monitoring site in the town of Alpine. The other 99 percent of San Diego County's 3 million residents breathe air that meets the 8-hour standard, but UCS still counts all of them as breathing air that violates the standard. Even about 65 percent of Los Angeles County's 10 million residents breathe air that complies with the 8-hour standard, as does everyone in the San Francisco Bay Area. Overall, about 30 percent of Californians live in areas that violate the federal 8-hour ozone standard—just one-third of what UCS claims.

Conclusion

In summarizing its case for harm from air pollution UCS states:

> *Numerous epidemiological studies tracking thousands of individuals have linked PM exposure to premature death as well as cardiovascular and respiratory illnesses. Similar studies have been carried out for exposure to ozone pollution…The health effects quantified in this report are based on peer-reviewed epidemiological studies used by both the EPA and CARB to evaluate the benefits of reducing air pollution. These studies establish a statistically significant relationship between exposure to PM and ozone and increased incidences of specific health endpoints…The uncertainty in these estimates is quantified by presenting results as both a mean estimate of the number of incidences and a range of estimates representing the 95 percent confidence interval.*[44]

This statement has the appearance of a weight-of-the-evidence scientific review, but it is misleading and disingenuous. First, UCS fails to mention the existence of a large body of evidence that contradicts its claims. Second, UCS implies that peer review provides quality assurance. But despite being peer reviewed, a large fraction of published epidemiology studies have little to do with reality.[45]

Third, UCS creates the false impression that the statistical certainty measure used—the 95 percent confidence interval—represents the real uncertainty in the estimates of air pollution's health effects derived in *Digging Up Trouble*. But the 95 percent confidence interval is a measure of real uncertainty only if the study subjects have been randomly selected and randomly assigned to pollution exposures, neither of which are the case in the studies UCS uses for its health effects claims. The 95 percent confidence interval isn't meaningful unless the biases created by non-random data, data mining, and publication bias have been removed.

At high enough concentrations, diesel exhaust can be an unpleasant and aggravating nuisance. But this is a far cry from UCS's accusation that more than a thousand people are killed each year or that hundreds of thousands suffer serious harm from construction-related air emissions. The weight of the evidence suggests that air pollution at current, historically low levels is a minor factor in people's health.[46]

According to its Web site, UCS "stands out among nonprofit organizations as the reliable source for independent scientific analysis."[47] UCS also leads a "scientific integrity" campaign devoted to opposing the manipulation of scientific research results for political ends. However, in *Digging Up Trouble* UCS selects and structures information to create the appearance of scientific support for its apparently predetermined conclusions about the health risks of air pollution from construction vehicles. The report fails to live up to UCS's own standards.

Joel Schwartz is a Visiting Fellow with the American Enterprise Institute.

Endnotes

[1] Union of Concerned Scientists, *Digging Up Trouble: The Health Risks of Construction Pollution in California* (Berkeley, CA: December 2006), http://www.ucsusa.org/assets/documents/clean_vehicles/Digging-up-Trouble.pdf.

[2] These percentages include only exhaust emissions. CARB also estimates that dust kicked up by "construction and demolition" accounted for about 5 percent of direct $PM_{2.5}$ emissions. Presumably some of these emissions are due to the movement of construction equipment on unpaved surfaces. It doesn't appear that UCS included these emissions in its estimates. 2005 is the year for which UCS estimated the health impacts of air pollution from construction equipment. California Air Resources Board, "Forecasted Emissions by Summary Category," last updated February 2, 2006, http://www.arb.ca.gov/app/emsinv/ccos/fcemssumcat_cc214.php; California Air Resources Board, "California Off-Road Diesel Fueled Equipment Inventory," October 2006, http://www.arb.ca.gov/msprog/ordiesel/documents/tier_distribution_table.pdf.

[3] Ibid.

[4] Air pollution has been dropping for as long as we've been measuring it—which means since the early or mid 1900s in some cases. California and national air pollution

emissions and ambient concentrations are at historic lows and continue to decline. For summary national trends in air pollution levels from 1980-2005, see www.epa.gov/airtrends and click on any of the pollutants for a trend graph. For California ozone and PM_{10} trend data, see http://www.arb.ca.gov/adam/cgi-bin/db2www/polltrendsb.d2w/start. For California air toxics (i.e., benzene, 1,3-butadiene) trend data, see http://www.arb.ca.gov/adam/toxics/toxics.html. Some areas, including Los Angeles and Pittsburgh, have data going back to the early- or mid-20th Century. See, for example, C. I. Davidson, "Air Pollution in Pittsburgh: A Historical Perspective," *Journal of the Air Pollution Control Association* 29 (1979): pp. 1035-41; J. H. Ludwig, G. B. Morgan and T. B. McMullen, "Trends in Urban Air Quality," *EOS* 51 (1970): pp. 468-75; H. W. Ellsaesser, "Trends in Air Pollution in the United States," in *The State of Humanity*, ed. J. L. Simon (Malden, MA: Blackwell, 1995), pp. 491-502.

[5] For more detailed discussions of popular portrayals of evidence on air pollution levels and health effects, see, for example, J. Schwartz, *Air Quality: Much Worse on Paper Than in Reality* (Washington, DC: American Enterprise Institute, May 2005), http://www.aei.org/docLib/20050602_EPOMay_Junenewg%282%29.pdf; J. Schwartz, *Air Pollution and Health: Do Popular Portrayals Reflect the Scientific Evidence?* (Washington, DC: American Enterprise Institute, May 2006), http://www.joelschwartz.com/pdfs/AirPoll_Health_EPO_0506.pdf; J. Schwartz, "Air Pollution: Why Is Public Perception So Different from Reality?" *Environmental Progress* 25 (2006): pp. 291-97.

[6] See note 4 for summary information on air pollution trends.

[7] UCS claims construction-related air pollution causes $9.14 billion per year in harm, of which $8.94 billion represents premature death.

[8] UCS doesn't make this explicit. However, *Digging Up Trouble* cites CARB's health-effects report on goods movement in California as the source for its air pollution death claims. CARB attributes 40 percent of premature deaths to nitrate PM specifically. The percentage breakdown for construction equipment might be a few percentage points higher or lower than for goods movement. There's no easy way to know for sure, because *Digging Up Trouble* provides only cursory information on the methodology used to derive its estimates, and does not provide any quantitative breakdowns of its results beyond the summary estimates of total health effects from all construction-related air pollution. In the absence of these details, I use CARB's goods-movement results as a reasonable ballpark breakdown of the fraction of all health effects contributed by the various components of construction-related air pollution. See California Air Resources Board, *Quantification of the Health Impacts and Economic Valuation of Air Pollution from Ports and Goods Movement in California* (Sacramento, CA: March 21, 2006), http://www.arb.ca.gov/planning/gmerp/march21plan/appendix_a.pdf, p. A-75.

[9] L. C. Green and S. R. Armstrong, "Particulate Matter in Ambient Air and Mortality: Toxicologic Perspectives," *Regulatory Toxicology and Pharmacology* 38 (2003): pp. 326-35; 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): pp. 317-26; 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): pp. 197-235; 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): pp. 189-96.

[10] See note 8 for how this estimate was derived.

[11] J. H. Seinfeld, "Urban Air Pollution: State of the Science," *Science* 243 (1989): pp. 745-52.

[12] Based on hourly ozone and NOx monitoring data for 1997–2001 downloaded from the California Air Resources Board's Web site, http://www.arb.ca.gov/aqd/aqdcd/aqdcddld.htm.

[13] C. L. Blanchard and S. J. Tannenbaum, "Differences between Weekday and Weekend Air Pollutant Levels in Southern California," *Journal of the Air & Waste Management Association* 53 (2003): pp. 816-28; E. M. Fujita, D. E. Campbell, B. Zielinska et al., "Diurnal and Weekday Variations in the Source Contributions of Ozone Precursors in California's South Coast Air Basin," *Journal of the Air & Waste Management Association* 53 (2003): pp. 844-63; R. A. Harley, L. C. Marr, J. K. Lehner et al., "Changes in Motor Vehicle Emissions on Diurnal to Decadal Time Scales and Effects on Atmospheric Composition," *Environmental Science and Technology* 39 (2005): pp. 5356-62.

[14] Blanchard and Tannenbaum, "Differences between Weekday and Weekend Air Pollutant Levels in Southern California"; C. L. Blanchard and S. J. Tannenbaum, "Weekday/Weekend Differences in Ambient Air Pollutant Concentrations in Atlanta and the Southeastern United States," *Journal of the Air & Waste Management Association* 56 (2006): pp. 271-84; E. M. Fujita, W. R. Stockwell, D. E. Campbell et al., "Evolution of the Magnitude and Spatial Extent of the Weekend Ozone Effect in California's South Coast Air Basin 1981-2000," *Journal of the Air & Waste Management Association* 53 (2003): pp. 864-75; Harley, Marr, Lehner et al., "Changes in Motor Vehicle Emissions on Diurnal to Decadal Time Scales and Effects on Atmospheric Composition"; D. R. Lawson, "The Weekend Effect–the Weekly Ambient Emissions Control Experiment," *Environmental Manager* (July 2003): pp. 17-25; L. C. Marr and R. A. Harley, "Modeling the Effect of Weekday-Weekend Differences in Motor Vehicle Emissions on Photochemical Air Pollution in Central California," *Environmental Science & Technology* 36 (2002): pp. 4099-106; L. C. Marr and R. A. Harley, "Spectral Analysis of Weekday-Weekend Differences in Ambient Ozone, Nitrogen Oxide, and Non-Methane Hydrocarbon Time Series in California," *Atmospheric Environment* 36 (2002): pp. 2327-35; B. K. Pun and C. Seigneur, "Day-of-Week Behavior of Atmospheric Ozone in Three U.S. Cities," *Journal of the Air & Waste Management Association* 53 (2003): pp. 789-801; R. Torres-Jardon and T. C. Keener, "Evaluation of Ozone-Nitrogen Oxides-Volatile

Organic Compound Sensitivity of Cincinnati, Ohio," *Journal of the Air & Waste Management Association* 56 (2006): pp. 322-33.

[15] H. Gong, Jr., C. Sioutas and W. S. Linn, "Controlled Exposures of Healthy and Asthmatic Volunteers to Concentrated Ambient Particles in Metropolitan Los Angeles" (Boston: Health Effects Institute, 2003); S. T. Holgate, T. Sandstrom, A. J. Frew et al., *Health Effects of Acute Exposure to Air Pollution. Part I: Healthy and Asthmatic Subjects Exposed to Diesel Exhaust* (Boston: Health Effects Institute, 2003).

[16] Y. Zhu, W. C. Hinds, S. Kim et al., "Concentration and Size Distribution of Ultrafine Particles near a Major Highway," *Journal of the Air and Waste Management Association* 52 (2002): pp. 1032-42.

[17] Green and Armstrong, "Particulate Matter in Ambient Air and Mortality: Toxicologic Perspectives"; S. H. Moolgavkar, "A Review and Critique of the EPA's Rationale for a Fine Particle Standard," *Regulatory Toxicology and Pharmacology* 42 (2005): pp. 123-44.

[18] Green and Armstrong, Ibid.

[19] Publication bias is a well-documented problem in a range of disciplines. See, for example, Victor M. Montori, Marek Smieja and Gordon H. Guyatt, "Publication Bias: A Brief Review for Clinicians," *Mayo Clinic Proceedings* 75 (2000): pp. 1284-88; Alison Thornton and Peter Lee, "Publication Bias in Meta-Analysis: Its Causes and Consequences," *Journal of Clinical Epidemiology* 53 (2000): pp. 207-16.

[20] 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.

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

[22] Anderson et al., *Meta-Analysis of Time-Series Studies and Panel Studies of Particulate Matter (PM) and Ozone*.

[23] For example, hormone replacement therapy and Vitamin A turned out not to reduce risk of cardiovascular disease, following a low-fat diet turned out not to reduce risk of heart disease or colorectal and breast cancer, and calcium supplements didn't reduce the risk of osteoporosis. S. A. Beresford, K. C. Johnson, C. Ritenbaugh et al., "Low-Fat Dietary Pattern and Risk of Colorectal Cancer: The Women's Health Initiative Randomized Controlled Dietary Modification Trial," *Journal of the American Medical Association* 295 (2006): pp. 643-54; B. V. Howard, L. Van Horn, J. Hsia et al., "Low-Fat Dietary Pattern and Risk of Cardiovascular Disease: The Women's Health Initiative Randomized Controlled Dietary Modification Trial," *Journal of the American Medical Association* 295 (2006): pp. 655-66; G. Kolata, "Big Study Finds No Clear Benefit of

Calcium Pills," *New York Times*, Feburary 16, 2006; Moolgavkar, "A Review and Critique of the EPA's Rationale for a Fine Particle Standard"; R. L. Prentice, B. Caan, R. T. Chlebowski et al., "Low-Fat Dietary Pattern and Risk of Invasive Breast Cancer: The Women's Health Initiative Randomized Controlled Dietary Modification Trial," *Journal of the American Medical Association* 295 (2006): pp. 629-42; G. D. Smith, "Reflections on the Limitations to Epidemiology," *Journal of Clinical Epidemiology* 54 (2001): pp. 325-31; G. Taubes, "Epidemiology Faces Its Limits," *Science* 269 (1995): pp. 164-69.

[24] Sharon Begley, "New Journals Bet 'Negative Results' Save Time, Money," *The Wall Street Journal*, September 15, 2006, p. B1, http://online.wsj.com/article/SB115827169620563571-email.html.

[25] J. P. Ioannidis, "Why Most Published Research Findings Are False," *PLoS Med* 2 (2005): e124, http://medicine.plosjournals.org/archive/1549-1676/2/8/pdf/10.1371_journal.pmed.0020124-L.pdf; Smith, "Reflections on the Limitations to Epidemiology"; Taubes, "Epidemiology Faces Its Limits."

[26] Anderson, Atkinson, Peacock et al., *Meta-Analysis of Time-Series Studies and Panel Studies of Particulate Matter (PM) and Ozone* (; M. L. Bell, F. Dominici and J. M. Samet, "A Meta-Analysis of Time-Series Studies of Ozone and Mortality with Comparison to the National Morbidity, Mortality, and Air Pollution Study," *Epidemiology* 16 (2005): pp. 436-45; K. Ito, "Associations of Particulate Matter Components with Daily Mortality and Morbidity in Detroit," in *Revised Analyses of Time-Series Studies of Air Pollution and Health* (Boston: Health Effects Institute, 2003); W. R. Keatinge and G. C. Donaldson, "Heat Acclimatization and Sunshine Cause False Indications of Mortality Due to Ozone," *Environmental Research* 100 (2006): pp. 387-93; G. Koop and L. Tole, "Measuring the Health Effects of Air Pollution: To What Extent Can We Really Say That People Are Dying from Bad Air?" *Journal of Environmental Economics and Management* 47 (2004): pp. 30-54.

[27] 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* (Boston: Health Effects Institute, July 2000); C. A. Pope, 3rd, 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): pp. 1132-41.

[28] Krewski, Burnett, Goldberg et al., *Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study*.

[29] J. E. Enstrom, "Fine Particulate Air Pollution and Total Mortality among Elderly Californians, 1973-2002," *Inhalation Toxicology* 17 (2005): pp. 803-16; F. W. Lipfert, H. M. Perry, J. P. Miller et al., "The Washington University-EPRI Veterans' Cohort Mortality Study," *Inhalation Toxicology* 12 (suppl. 4) (2000): pp. 41-73.

[30] M. L. Bell, A. McDermott, S. L. Zeger et al., "Ozone and Short-Term Mortality in 95 US Urban Communities, pp. 1987-2000," *Journal of the American Medical Association* 292 (2004): pp. 2372-8; F. Dominici, A. McDermott, M. Daniels et al., *Revised Analyses of the National Morbidity, Mortality, and Air Pollution Study* (Boston: Health Effects Institute, May 2003).

[31] UCS, p. 7.

[32] See UCS, p. 25. UCS says its health effects estimates are derived from health studies cited in the following reports from CARB and EPA: California Air Resources Board, *Appendix A. Quantification of the Health Impacts and Economic Valuation of Air Pollution from Ports and Goods Movement in California* (Sacramento: March 21, 2006), http://www.arb.ca.gov/planning/gmerp/march21plan/appendix_a.pdf; Environmental Protection Agency, *Final Regulatory Analysis: Control of Emissions from Nonroad Diesel Engines* (Washington, DC: May 2004), http://www.epa.gov/nonroad-diesel/2004fr/420r04007a.pdf.

[33] CARB's report does not provide these percentages explicitly. Instead, in one part of its staff report CARB estimates the number of incidences of various health effects avoided by reducing ozone. Another part of the report provides estimates of the total number of incidences of each health effects. Dividing the former by the latter gives the fraction of health effects avoided by reducing ozone. I demonstrate this in 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. For CARB's estimates, see 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.

[34] Once again, EPA does not provide explicit percentage changes, but the percentage changes can be calculated from data provided in a journal article by EPA's scientists. See 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): pp. 73-82; Schwartz, *Rethinking the California Air Resources Board's Ozone Standards*.

[35] 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: California Air Resources Board, July 2002).

[36] UCS, p. 9.

[37] 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): pp. 1859-67.

[38] For data on asthma emergency room visits and hospitalizations by month, see, for example, Spokane Regional Health District, *Asthma in Spokane County* (Spokane, WA: April 2002), http://www.srhd.org/information/pubs/pdf/factsheets/AsthmaInSpokaneCounty.pdf; J. K. 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.

[39] UCS, p. 9.

[40] California Air Resources Board, *Appendix A. Quantification of the Health Impacts and Economic Valuation of Air Pollution from Ports and Goods Movement in California*.

[41] 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): pp. 43-54.

[42] K. Berhane and D. C. Thomas, "A Two-Stage Model for Multiple Time Series Data of Counts," *Biostatistics* 3 (2002): pp. 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): pp. 1103-15.

[43] There are other reasons to conclude that the claim of a connection between ozone and school absences is not credible. For details, see pp. 28-30 in Schwartz, *Rethinking the California Air Resources Board's Ozone Standards*.

[44] UCS, p. 25.

[45] Ioannidis, "Why Most Published Research Findings Are False"; Smith, "Reflections on the Limitations to Epidemiology"; Taubes, "Epidemiology Faces Its Limits"; Begley, "New Journals Bet 'Negative Results' Save Time, Money."

[46] See note 4 for summary information on air pollution trends.

[47] UCS, "About UCS," http://www.ucsusa.org/ucs/about/.