Comments on EPA's Proposal to Adopt a More Stringent Ozone Standard

Comments Submitted to Docket No. EPA-HQ-OAR-2005-0172

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Introduction

On July 11, 2007, the Environmental Protection Agency (EPA) proposed a more stringent national standard for ambient ozone levels (the "Proposed Rule").[1] EPA is considering a new standard somewhere in the range of 0.060 to 0.080 parts per million (ppm), with a preferred value in the range of 0.070-0.075 ppm.

EPA relies mainly on two major types of human health effects studies in an attempt to justify a more stringent ozone standard: observational epidemiology studies and controlled laboratory studies with human volunteers. According to EPA, both types of studies provide evidence that ozone has adverse health effects at levels below the current 8-hour ozone standard of 0.085 ppm. EPA is mistaken. In its proposed rule, presentations, criteria documents, staff reports, and other paperwork, EPA creates a misleading appearance that there is a vast body of robust and consistent evidence for its claims of harm from low-level ozone. As I show below, the weight of the evidence suggests just the opposite. The current 8-hour ozone standard is more than stringent enough to protect Americans' health "with an adequate margin of safety."

In my main comments, I focus on two overarching issues: First, I present evidence that observational epidemiology studies generate false indications of risk through data mining and publication bias. Observational studies tend to confirm the preconceptions and prejudices of the researchers, rather than provide realistic information on health effects. EPA cites hundreds of air pollution epidemiology studies as "evidence" of harm from ozone. But implementing an invalid methodology over and over again doesn't improve its validity.

As a result of the serious weaknesses of observational epidemiology studies, this type of evidence should be considered invalid even without going through a detailed assessment of the results of each individual study. Nevertheless, in appendices to these comments I provide a detailed assessment of many of the studies EPA cites in support of a tougher ozone standard and discuss weaknesses and contradictory evidence that EPA chose to omit from its documentation. I show that even if we assume that observational studies are telling us something real about the health effects of ozone, the story they tell is very different from the one EPA creates through its selective choice and structuring of the available evidence.

Second, I demonstrate that, contrary to EPA's claims, laboratory studies with human volunteers show that the current 8-hour ozone standard is already stringent enough to protect Americans' health.

As a result, the weight of the evidence suggests that the current ozone standard is more than stringent enough to protect Americans' health. EPA's proposed rule for a tougher ozone standard is unjustified and should be withdrawn.

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[1] Federal Register, July 11, 2007, pp. 37818-37919.

Observational Epidemiology Studies Give Spurious Results

In making its case for a tougher ozone standard, EPA notes that "The Criteria Document prepared for this review [of the ozone standard] emphasizes a large number of epidemiological studies published since the last review…" Indeed, the Criteria Document (CD) and Staff Report cite hundreds of epidemiological studies that EPA claims provide robust and consistent support for the claim that ozone causes serious harm, even at levels below the current 8-hour standard. EPA's PowerPoint presentation on the Proposed Rule highlights the "Large number of new epidemiological studies, including new multi-city studies, [that] strengthen EPA's confidence in the links between ozone exposure and health effects. New studies link ozone exposure to important new health effects, including mortality, increased asthma medication use, school absenteeism, and cardiac-related effects."[2] What EPA avoids discussing is that these epidemiological studies are all of a type known as an "observational" study and that observational studies have been shown to give spurious results.

Observational epidemiology 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. 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.

Unlike controlled clinical or 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 an asthma attack, 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.

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. Even more importantly, incentives for publication bias and data mining cause an exaggeration of 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

[2] EPA, June 2007 Proposal to Revise the National Ambient Air Quality Standards for Ground-level Ozone, http://www.epa.gov/air/ozonepollution/pdfs/20070627slides.pdf.

having lots of "dials" or "knobs" that researchers can turn in order to "tune" the statistical model to fit the observations. Researchers tend to turn these knobs and dials in ways that maximize the effects they "expect" 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.[3] 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*.[4]

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. <u>This model selection problem is exacerbated by the common practice of screening multiple analyses and then selectively reporting only a few important results</u>*.[5]
> (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*.[6]

Publication bias and data mining are serious problems not only in air pollution epidemiology but in 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, which do not suffer from the biases inherent in observational studies.[7]

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[3] 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): 1284-88; Alison Thornton and Peter Lee, "Publication Bias in Meta-Analysis: Its Causes and Consequences," *Journal of Clinical Epidemiology* 53 (2000): 207-16.

[4] 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.

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

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

[7] 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

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.[8] 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 small, as they are for air pollution.[9] A number of studies have also provided direct evidence that observational studies of air pollution and health are generating false indications of risk as a result of data mining and publication bias.[10]

For obvious ethical and practical reasons, the existence of serious health effects due to low-level ozone (and other air pollution) exposures can't be evaluated in randomized controlled trials with humans, so all we have to go on are observational studies. This is advantageous for EPA and other advocates of expanded and more stringent regulation, because it means that the observational studies on which their power depends are unlikely to be tested for real-world validity. However, since the vast majority of observational studies have been overturned when tested in randomized trials, the prudent course is to conclude that air pollution epidemiology studies are no more valid than other observational studies. Indeed, there is reason to believe that observational air pollution studies are even *less likely* to be valid. The chance that an observational study's results are spurious *increases* as the magnitude of the putative health effect *decreases*.[11] The

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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): 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): 655-66; G. Kolata, "Big Study Finds No Clear Benefit of Calcium Pills," *New York Times*, Feburary 16, 2006; S. H. Moolgavkar, "A Review and Critique of the EPA's Rationale for a Fine Particle Standard," *Regulatory Toxicology and Pharmacology* 42 (2005): 123-44; 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): 629-42; G. D. Smith, "Reflections on the Limitations to Epidemiology," *Journal of Clinical Epidemiology* 54 (2001): 325-31; G. Taubes, "Epidemiology Faces Its Limits," *Science* 269 (1995): 164-69.

[8] 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.

[9] J. P. Ioannidis, "Why Most Published Research Findings Are False," *PLoS Med* 2 (2005): e124; Smith, "Reflections on the Limitations to Epidemiology;" Taubes, "Epidemiology Faces Its Limits."

[10] 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): 436-45; K. Ito, "Assocations 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): 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): 30-54.

[11] Ioannidis, "Why Most Published Research Findings Are False."

putative risks of current levels of air pollution are tiny compared to the putative health risks assessed in medical intervention studies.

EPA cites the large number of observational epidemiology studies claiming harmful effects of low-level ozone as evidence that the harm is real. But implementing an invalid methodology over and over again doesn't improve its validity. Rather, EPA should acknowledge that observational studies are not an appropriate basis for assessing the health effects ozone at or below current ambient levels.

Laboratory Studies

EPA claims laboratory studies with human volunteers provide direct evidence that ozone causes adverse effects at levels below the current 0.085 ppm standard. EPA places special emphasis on studies by Adams (2002, 2006)[12] because "they are the only available controlled exposure human studies that examine respiratory effects associated with prolonged O_3 exposures at levels below 0.080 ppm."

In these studies, healthy college students were exposed to ozone at various concentrations for 6.6 hours while exercising, and their lung function and subjective symptoms were measured several times during the exposure period. EPA reports that after 6.6 hours, 2 of 30 subjects in the study experienced temporary lung-function reductions of 10 percent or more (as measured by forced expiratory volume in one second (FEV_1)) when exposed to ozone at 0.060 ppm. Five of 30 subjects experienced at least a 10 percent decline in FEV_1 at an ozone concentration of 0.080 ppm.[13] Based on these results, EPA concludes that the current standard of 0.085 ppm does not sufficiently protect people from ozone.

This conclusion is unwarranted, because of the well known difference between ozone concentrations measured at the fixed ambient monitoring stations used to determine compliance with federal ozone standards ("ambient concentrations") and the ozone concentration in the air people actually inhale ("personal exposure").

Comparisons of ambient concentrations with personal ozone exposures measured by wearable monitors show that personal exposures are much lower than ambient concentrations. This is true even when comparing only *outdoor personal exposures* to *outdoor ambient levels*. Thus, for example, when the ambient ozone concentration is 0.08 ppm, personal exposures while outdoors are about 40 to 60 percent *lower* than the ambient level.[14] Or to put it the opposite way, for any given personal exposure, the ambient concentration is 1.67 to 2.5 times greater.

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[12] W. C. Adams, "Comparison of Chamber and Face-Mask 6.6 Hour Exposures to Ozone on Pulmonary Function and Symptom Responses," *Inhalation Toxicology* 14 (2002): 745-64; W. C. Adams, "Comparison of Chamber 6.6-hour Exposures to 0.04–0.08 ppm Ozone via Square-Wave and Triangular Profiles on Pulmonary Responses," *Inhalation Toxicology* 18 (2006): 127-36.

[13] Proposed Rule, p. 37828. Adams reported only group-mean results in the two journal articles. However, Adams provided EPA with data on each subject's response to ozone, which EPA analyzed for inclusion in its regulatory documents.

[14] See, for example, R. J. Delfino, B. D. Coate, R. S. Zeiger et al., "Daily Asthma Severity in Relation to Personal Ozone Exposure and Outdoor Fungal Spores," *American Journal of Respiratory and Critical Care*

This difference is crucial, because the ozone concentrations used in laboratory studies with human volunteers represent *personal exposures*. As a result, EPA is comparing apples to oranges when it makes believe the effects of, say, 0.060 ppm ozone in the laboratory provides information on the health effects of 0.060 ppm ozone measured at an ambient compliance monitor. In fact, an ozone concentration of 0.060 ppm in the laboratory represents an ambient-monitor concentration of at least 0.100 ppm—a level far greater than the current 8-hour standard of 0.085 ppm.

Table 1, below, translates the personal ozone exposures used in Adams (2006) into equivalent concentrations at ambient compliance monitors. Adams (2006) used two types of exposure patterns and four different average ozone concentrations. The "square" pattern is a constant ozone concentration for the 6.6-hour exposure period. In the "triangular" pattern, ozone rises to a peak and then falls. Because ambient-monitor levels are at least 1.67 times personal exposures, Table 1 uses a conversion factor of 1.67 to go from the personal exposure levels in Adams (2006) to equivalent ambient levels. The table gives both the average and peak level for each exposure pattern.

After converting to ambient levels, we can see that the ozone exposures in Adams (2006) were actually much greater than EPA claimed. For example, to get a personal exposure of 0.060 ppm, the ambient concentration would need to be at least 0.100 ppm. Both the 0.06 ppm and 0.08 ppm laboratory exposures are thus substantially greater than the current 8-hour, 0.085 ppm standard (marked in bold in the table). Furthermore, the triangular 0.060 ppm exposure, and both of the 0.080 ppm exposures exceeded even the old 1-hour, 0.125 ppm standard (italicized entries). Contrary to EPA's assertions, the Adams studies did not in fact test whether ozone has adverse effects at levels below 0.085 ppm.

Medicine 154 (1996): 633-41; A. S. Geyh, J. Xue, H. Ozkaynak et al., "The Harvard Southern California Chronic Ozone Exposure Study: Assessing Ozone Exposure of Grade-School-Age Children in Two Southern California Communities," *Environmental Health Perspectives* 108 (2000): 265-70; T. Johnson, K. Clark, K. Anderson et al., "A Pilot Study of Los Angeles Personal Ozone Exposures During Scripted Activities," Measurement of Toxic and Related Air Pollutants, Research Triangle Park, NC, Air and Waste Management Association, May 7-9, 1996; K. Lee, W. J. Parkhurst, J. Xue et al., "Outdoor/Indoor/Personal Ozone Exposures of Children in Nashville, Tennessee," *Journal of the Air and Waste Management Association* 54 (2004): 352-9; L. J. Liu, R. Delfino and P. Koutrakis, "Ozone Exposure Assessment in a Southern California Community," *Environmental Health Perspectives* 105 (1997): 58-65; M. S. O'Neill, M. Ramirez-Aguilar, F. Meneses-Gonzalez et al., "Ozone Exposure among Mexico City Outdoor Workers," *Journal of the Air and Waste Management Association* 53 (2003): 339-46.

Table 1. Comparison of personal ozone exposures used in Adams (2006) with equivalent ambient-monitor concentrations

Adams (2006) personal ozone exposure protocol			Equivalent ambient-monitor ozone concentration	
Exposure Pattern	6.6-hour Average Personal Ozone Exposure Concentration	Peak-Hour Personal Ozone Exposure Concentration	6.6-hour Average Concentration	Peak-Hour Concentration
Filtered Air	0.000	0.000	0.000	0.000
Triangular	0.040	0.050	0.066	0.084
Square	0.060	0.060	**0.100**	0.100
Triangular	0.060	0.090	**0.100**	*0.150*
Square	0.080	0.080	**0.134**	*0.134*
Triangular	0.080	0.150	**0.133**	*0.250*

Notes: All values are in parts per million (ppm). Bold entries signify ozone exposures that exceeded the current 8-hour, 0.085 ppm ambient standard. Italicized entries signify exposures that exceeded the 1-hour, 0.125 ppm standard. A "square" exposure pattern means that subjects breathed a constant ozone concentration for the 6.6-hour study. A "triangular" exposure pattern means subjects breathed ozone that rose to a peak during the first half of the exposure period and then fell during the second half.

EPA is thus engaging in some sleight of hand when it claims laboratory studies show that ozone has adverse effects at concentrations as low as 0.060 ppm. The 0.060 ppm that EPA refers to is a *personal exposure* but EPA uses it as if it were an *ambient-monitor concentration*. If anything, Adams (2006) shows that even ozone levels substantially greater than the current 8-hour standard have little effect on people's lung function. A study with personal exposures closer to the current 8-hour standard (a laboratory exposure of 0.051 ppm would be equivalent to the current ambient 8-hour standard of 0.085 ppm) is unlikely to have any effect, suggesting that the current standard is more than stringent enough to protect people's health.

EPA is aware of the difference between ozone concentrations measured at ambient monitors and actual personal exposure concentrations. In its Proposed Rule, EPA notes that "using ambient concentrations to determine exposure generally overestimates true personal O_3 exposures by approximately 2- to 4-fold in available studies."[15] But EPA brings up this issue in the context of observational epidemiology studies, while ignoring

[15] The reasons for this difference include not only the fact that actual personal ozone exposure while outdoors is lower than ambient monitor measurements by at least 40 percent, as described above, but also because people spend most of their time indoors.

the implications of this difference between ambient concentration and personal exposure for the interpretation of laboratory studies with human volunteers.

Conclusion

EPA's case for a more stringent ozone standard rests on false premises. First, observational epidemiology studies create false indications of risk where no risk in fact exists. Second, even the lowest ozone exposures used in laboratory studies are higher than the current 8-hour ozone standard, yet EPA mistakenly claims these studies assess exposures below the current 8-hour standard. Nevertheless even these relatively high laboratory exposures show little evidence of harm from ozone. The attached appendices provide additional evidence that the current 8-hour standard of 0.085 ppm protects public health with room to spare. EPA's proposal for a more stringent 8-hour ozone standard is therefore unjustified and should be withdrawn.[16]

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Appendices

I. Joel Schwartz. Comments on EPA's Ozone Criteria Document; 2nd External Review Draft. December 2, 2005.

II. Joel Schwartz. Rethinking the California Air Resources Board's Ozone Standards. September 12, 2005.

III. Joel Schwartz. Air Pollution and Health: Do Popular Portrayals Reflect the Scientific Evidence? April 2006.

Appendix I

Comments on EPA's Ozone Criteria Document;
2nd External Review Draft

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December 2, 2005

Fred Butterfield
Designated Federal Officer
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Dear Mr. Butterfield:

Thank you for the opportunity to comment on the 2nd External Review Draft of the Environmental Protection Agency's Ozone Criteria Document (CD). While the ozone CD includes a great deal of information, it nevertheless provides a misleading and incomplete view of ozone's health effects and the potential benefits of additional ozone reductions. The comments below detail my specific concerns.

Maximum Health Benefits of Ozone Reductions

While the CD includes hundreds of pages of information on ozone's health effects, nowhere does it summarize in practical terms, the health improvements EPA believes will occur if ozone is reduced from current levels. The CD and Staff Report should include a table in the Executive Summary of each document listing EPA's best estimates of the percentage reduction in various health effects (e.g., asthma emergency room visits, respiratory hospital admissions, premature deaths) that might result from reducing ozone down to various levels, including down to the level of the policy-relevant background (PRB).

Table 1 provides an example, based on estimates from the California Air Resources Board (CARB) of the benefits of attaining the current federal 8-hour standard of 0.085 ppm (4th highest annual reading), and of attaining a standard of 0.070 ppm (no exceedances allowed), which is the standard CARB adopted for California.[1] The estimates are based on ozone levels in California during 2001-2003.

[1] For details, see 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.

Table 1. Percent of Health Effects Avoided by Going from Recent California Ozone Levels Down to the Federal 8-Hour Standard and to the California 8-Hour Standard

Health Effect	Attainment of Federal 8-Hour Standard (0.085 ppm; 4th highest)	Incremental Benefits of California 8-Hour Standard (0.070 ppm; no exceedances)	Total Reduction in Cases
Mortality	0.14%	0.13%	0.27%
Respiratory Hospital Admissions	0.60%	0.55%	1.15%
Asthma ER Visits	0.92%	0.84%	1.76%

CARB estimated the number of cases of each health condition avoided by reducing ozone, and also provided the estimated baseline rate of each health condition in California's population. Since we know California's total population, I estimated the percent reduction in the total number of cases for each health outcome.

Because reducing ozone on peak days at peak sites in an air basin also means reducing ozone on all other days and sites in that air basin, most of the health benefits accrue from reductions in ozone levels that already comply with the given standard. The table therefore assumes that benefits accrue until ozone is reduced to 0.04 ppm, which is CARB's (somewhat arbitrary) choice for the PRB. Based on CARB's estimates, only 20 percent of the benefits listed in the table are due to reducing ozone down to the level of the given standard.

The California 8-hour ozone standard of 0.070 ppm, not to be exceeded, should probably be considered as requiring ozone to be reduced to the PRB. Even if the average daily 8-hour-average PRB is lower than 0.070 ppm, the standard is based on peak values, so the relevant comparison is with the PRB on the day with the highest PRB ozone level of the year. Given natural biogenic emissions within California, and ozone and ozone precursors transported into the state from elsewhere, some parts of the state would probably exceed the California standard at least once every few years even without any human-caused emissions from within the U.S. Adding in other emissions that could reasonably considered part of the PRB, such biogenic emissions from agriculture, and the small amounts of VOC and NO emitted by human bodies, would make it still more likely that the California standard is near or perhaps below the PRB in some areas of the state.

If so, then the estimates in Table 1, with appropriate caveats, can be considered the maximum health benefits of eliminating all human-caused ozone. These caveats include at least the following: (1) the assumption that CARB's estimates of ozone dose-responses for various health effects reflect the weight of the evidence from the scientific literature, and (2) that CARB used a defensible rollback method for estimating ozone reductions that would occur on non-peak days due to efforts to reduce ozone on peak days. If anything, CARB erred on the side of overstating the both ozone reductions and the health

benefits of each increment of ozone reductions, so the values in Table 1 should be considered upper limits on health benefits.

A recent study by EPA scientists also provides estimates of health improvements from reducing ozone.[2] In this case, the estimates were for the health benefits of reducing ozone only down to the federal 8-hour standard, with no additional benefits assumed for reductions in ozone below the standard. Just as in CARB's case, the EPA study provides only the estimated number of cases avoided, but also provides the estimated baseline rate of each health outcome in the population. Combined with a knowledge of the total relevant population, I calculated the percent reduction in each health outcome, as shown in Table 2. Once again, these results are based on ozone levels during 2001-2003. The benefits would probably be about a factor of 5 greater if benefits continue to accrue as ozone is reduced below the federal 8-hour standard.

Table 2. Percent of Health Effects Avoided by Going from Recent National Ozone Levels Down to the Federal 8-Hour Standard

Health Effect	**Attainment of Federal 8-Hour Standard (0.085 ppm; 4th highest)**
Premature Mortality	0.03%
Respiratory Hospital Admissions, Adults	0.03%
Respiratory Hospital Admissions, Children	0.05%
Asthma ED Visits	0.02%

Both EPA's and CARB's estimates show that the total effects of ozone on public health are small. The true benefits are even smaller than EPA's and CARB's estimates would lead one to believe, because EPA and CARB selected the evidence in such a way as to bias upward the estimated health effects of ozone.[3] Regardless, whatever benefits EPA believes will accrue from reducing ozone, these benefits should be clearly listed up front as a percentage reduction from the baseline level of each health outcome. The public must know the extent to which reducing ozone can reduce the total burden of various health effects suffered by Americans, if there is to be a reasoned and informed discussion of ozone policy.

[2] B. J. Hubbell, A. Hallberg, D. R. McCubbin et al., "Health-Related Benefits of Attaining the 8-Hr Ozone Standard," *Environmental Health Perspectives* 113 (2005): 73-82.

[3] I discuss some of the reasons for this below, and also in Schwartz, *Rethinking the California Air Resources Board's Ozone Standards*.

Reducing ground-level ozone also will cause small increases in human exposure to solar ultraviolet (UV) radiation. I will have more to say about the CD's and Staff Report's treatment of this issue below. Here I want to stress that any discussion of the total effects of ground-level ozone reductions would be incomplete and misleading if it did not include an estimate of the negative health effects of ground-level ozone reductions. In this case, the appropriate comparison is between the absolute number of health outcomes prevented (e.g., premature mortality, respiratory hospital admissions) by ozone reductions and the absolute number of health outcomes caused (e.g., premature mortality, non-fatal skin cancers, cataracts) by ozone reductions.

The effects of the costs of reducing ozone on Americans' health and welfare are not required to be addressed by EPA in its CD. However, these costs and their ensuing health and welfare effects are real, and EPA would better serve the public by acknowledging these costs in the CD.[4]

Selective Characterization of Evidence

Although the CD includes many caveats in its discussions of the evidence on ozone's health effects, the CD overall selectively emphasizes studies and portions of studies reporting harmful ozone effects, while downplaying studies reporting no effects or apparently protective ozone effects. Likewise, the CD is quick to emphasize weaknesses in studies that report little or no harmful effect from ozone, while ignoring weaknesses in studies reporting harmful effects. Summary sections of the CD often draw conclusions that are at odds with the detailed evidence presented in more technical and detailed sections of the CD.

For example, in a summary in Chapter 8, the CD claims ozone effect sizes are relatively consistent across studies (p. 8-56). However, this claim is based on pooling of results across cities and/or studies, and masks the large heterogeneity of the results between individual cities and even the same city across individual studies. For example, NMMAPS reported a range of a -5% to +16% increase in mortality per 10 ppb increase in 24-hour ozone across the 95 cities in the study. Higher ozone was associated with reduced mortality in nearly 40 percent of the cities in the study.

However, the CD focuses only on the pooled results in its summary of the epidemiologic literature on ozone and mortality.[5] The CD draws conclusions based on the pooled results, rather than the individual city results, creating an appearance of consistency that does not in fact exist. The city-by-city data from NMMAPS and other studies cited in the CD demonstrate the huge and biologically implausible range of apparent ozone effects on mortality, from very protective to very harmful.

The CD also fails to note that the pooled result in NMMAPS is sensitive to a few outlier cities. Moolgavkar (2002, 2005), has shown that the NMMAPS pooled PM_{10} mortality association becomes statistically insignificant when just two or three outlier cities are

[4] For more on this, see, for example, Ibid.

[5] See Figure 3 in M. L. Bell, A. McDermott, S. L. Zeger et al., "Ozone and Short-Term Mortality in 95 US Urban Communities, 1987-2000," *Journal of the American Medical Association* 292 (2004): 2372-8.

removed from the analysis.[6] Examination of Figure 3 in Bell et al. (2005) suggests that one extreme outlier city and two or three more moderate outliers are driving the positive ozone result as well. The CD demonstrates the great heterogeneity of ozone associations in the technical sections of the document, but the summary sections draw conclusions about consistency that are at odds with this evidence.

The CD makes a few mentions of publication bias and model-selection bias in ozone epidemiology studies, but these concerns seem to have had little effect on the CD's actual use of evidence and conclusions. For example, the CD cites three recent EPA-commissioned meta-analyses[7] in support of the conclusion that daily ozone fluctuations are increasing daily mortality, stating: "These three studies, along with the earlier meta-analyses, provide strong evidence that O_3 is associated with mortality" (p. 7-84). This claim ignores the degree to which publication bias inflated the ozone effect estimates in these studies. For example, Bell et al. (2005) presented evidence that publication bias may have inflated the meta-analytic ozone effect estimate by more than a factor of 3—a fact not mentioned in the CD.

The CD claims that the consistency of the results lends weight to their conclusions. But this consistency is likely do to the three studies sharing the same biases, rather than to an underlying relationship of the results to real-world health effects. As a commentary accompanying the meta-analyses concluded: "In the absence of NMMAPS or other multisite analyses, some observers might have taken the agreement of the meta-analyses as confirmation that the meta-analytic method was reliable. However, if our observational methods are all subject to the same biases, as meta-analyses are when they are derived from the same pool of studies, the agreement criterion is testing a narrow range of assumptions."[8]

The CD's cursory treatment of publication bias also points up the selective way in which the CD marshalls evidence. The CD uses the Bell et al. (2004) NMMAPS results as evidence of an ozone mortality effect, but discounts these same NMMAPS results when they provide inconvenient evidence about the effect of publication bias in inflating the meta-analytic ozone-mortality estimates.

As with the effect of publication bias, the CD mentions, but fails to adequately account for the degree to which model selection bias inflates ozone effect estimates. Koop and Tole used Bayesian Model Averaging (BMA) to conclude, based on data for Toronto,

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[6] S. H. Moolgavkar, *Review of Chapter 8 of the Criteria Document for Particulate Matter (Comments Submitted to EPA)* (2002); S. H. Moolgavkar, "A Review and Critique of the Epa's Rationale for a Fine Particle Standard," *Regulatory Toxicology and Pharmacology* 42 (2005): 123-44.

[7] 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): 436-45; K. Ito, S. F. De Leon and M. Lippmann, "Associations between Ozone and Daily Mortality: Analysis and Meta-Analysis," *Epidemiology* 16 (2005): 446-57; J. I. Levy, S. M. Chemerynski and J. A. Sarnat, "Ozone Exposure and Mortality: An Empiric Bayes Metaregression Analysis," *Epidemiology* 16 (2005): 458-68.

[8] S. N. Goodman, "The Methodologic Ozone Effect," *Epidemiology* 16 (2005): 430–35.

that ozone is unlikely to be associated with daily mortality.[9] The CD summarily dismisses this research with a few sentences about BMA's limitations in the introduction to Chapter 7, and does not consider the technique further. Koop and Tole (2004) is not mentioned at all in the Staff Report.

Yet the problem of model selection bias is becoming widely recognized in air pollution epidemiology, and Koop and Tole (2004) is one of the few efforts to systematically address the issue. For example, the Health Effects Institute special panel that reanalyzed the GAM time series studies concluded that various model selection choices may "introduce an element of uncertainty that has not been widely appreciated previously."[10] Likewise, Ito (2003), in the same report, concluded:[11]

> "Weather model specification and the extent of temporal smoothing are not the only factors that can change pollution [Relative Risk] estimates. Others may include the location of monitors, choice of lags, and consideration of distributed lags. These factors can cause differences that vary by up to a factor of two in estimated pollution coefficients."

These problems are compounded by the selective publication of larger and more statistically significant effects. Lumley and Sheppard (2003) cautioned:

> "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."[12]

In fact, Koop and Tole is not the only paper, and BMA is not the only method of demonstrating the effects of model-selection bias. Ito (2003) estimated 1,220 separate air pollution-mortality models for Detroit and substantial fraction suggested a "protective" effect of air pollution on health.

A new study shows that changes in adjustment for weather can cause the apparent effect of ozone on short-term mortality to disappear. When Keatinge and Donaldson (2005) allowed in their model for cumulative effects of heat stress over several days, as well as

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[9] 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): 30-54.

[10] Health Effects Institute, *Revised Analyses of Time-Series Studies of Air Pollution and Health* (Boston: May 2003).

[11] K. Ito, "Assocations of Particulate Matter Components with Daily Mortality and Morbidity in Detroit," in *Revised Analyses of Time-Series Studies of Air Pollution and Health*, ed. (Boston: Health Effects Institute, 2003).

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

the additional effects of direct sunshine, which adds to heat stress, the association of ozone with mortality was reduced by 90 percent and became statistically insignificant.[13]

The CD's density plots indirectly show that consideration of publication and model-selection bias would greatly reduce the health effects attributed to ozone. For example, the density plot for mortality on page 7-128 has 25 percent of the probability on the side of a protective effect for ozone. But for multi-city studies, the chart relies on pooled results, rather than individual city results. For example, NMMAPS includes 95 city results, nearly 40 percent of which suggested a protective effect of ozone. Entering results for individual cities would increase the amount of probably on the side of protective ozone effects. Furthermore, the density chart relies only on published point-estimate studies, and therefore suffers from publication bias (only partially accounted for by having a few multi-city studies) and model selection bias (not accounted for at all). Accounting for these effects would push still more of the probability toward negative (that is, protective) ozone effects.

The CD's conclusion of robustness and consistency of ozone associations with mortality and other health endpoints is mistaken, and its presentation of the evidence is misleading. EPA should revise the CD to reflect the lack of consistency and biologic implausibility of the epidemiological results.

Selective and Inaccurate Characterizations of Studies

I discussed above the CD's general problem of mischaracterizing evidence. Here I point out some additional cases in which the CD mischaracterizes specific studies, creating a bias toward assuming greater air pollution health effects than the actual results of the studies would suggest.

Children's Health Study Asthma Results: The California Children's Health Study (CHS) assessed the risk of developing asthma due to air pollution in a cohort of 3,535 children with a five-year follow-up.[14] According to the CD, "Asthma risk was not higher for residents of the six high-O_3 communities versus residents of the six low-O_3 communities" (p. 7-109). The staff report makes a similar claim (p. 3-22). These claims are mistaken. The risk of asthma was 30 percent lower in the six high-ozone communities, relative to the six low-ozone communities in the study.[15]

The CD notes that asthma risk was 3.3 times greater for children in high-ozone communities playing 3 or more team sports (8 percent of the children), though this result was based on a small sample. This means the risk of developing asthma must have been 50 percent lower for the other 92 percent of children in the study. When the 12 communities were divided into tertiles, increased asthma risk was reported for only the 4 highest ozone communities. These 4 high-ozone communities—all in the eastern portions of the South Coast Air Basin (the Los Angeles metro area)—have by far the highest

[13] W. R. Keatinge and G. C. Donaldson, "Heat Acclimatization and Sunshine Cause False Indications of Mortality Due to Ozone," *Environmental Research* (2005): in press.

[14] R. McConnell, K. Berhane, F. Gilliland et al., "Asthma in Exercising Children Exposed to Ozone: A Cohort Study," *Lancet* 359 (2002): 386-91.

[15] Based on 1-hour ozone levels. Based on 8-hour ozone levels, risk of asthma was 20% lower and the top of the 95% confidence interval for relative risk was 1.0).

ozone levels in the country. The study was based on ozone levels during 1994-97, when these areas violated the 1-hour ozone standard dozens of times per year. The rest of the U.S. has ozone levels typical of the medium- and low-ozone areas of the Children's Health Study, for which there was no increase in risk of developing asthma, even in very active children. Thus, this study suggests the federal 1-hour ozone standard is more than protective against the development of asthma.

If the higher asthma risk with higher ozone for very-active children is to be taken as causal, then there is no justification for not taking the lower overall asthma risk as also causal. If so, there are two conclusions that the CD and Staff Report should draw: First, overall, higher ozone levels reduce the risk of developing asthma. Second, the federal 1-hour and 8-hour ozone standards protect against the development of asthma, with a large margin of safety, even in the most physically active children. The CD and Staff Report should not create the impression that a more stringent ozone standard would reduce children's risk of developing asthma.

Multi-City Study of Ozone and Use of Asthma Medication. The CD claims "the strong evidence from the large multicities [sic] study by Mortimer et al. (2002)" (p. 8-44) shows that ozone is associated with increased medication use.[16] But the evidence from this study is not strong. The ozone effect was statistically significant only in a single-pollutant model. It became statistically insignificant when any other pollutant was added as a confounder. The CD creates the false impression that other pollutants had little confounding effect on the results: "In multipollutant models, the O_3 effect was shown to be *slightly* diminished" (p. 7-45; emphasis added). In fact, the ozone effect dropped by 40 percent when NO_2 was added to the model, and dropped to zero when NO_2, SO_2, and PM_{10} were added.[17]

CARB/Kaiser Central Valley Study. This time-series study reported a statistically significant decrease in acute health effects with higher ozone levels.[18] The CD does not mention this study.

EPRI-Veterans Cohort study: The CD states "Lipfert et al. (2000b, 2003) reported positive effects on all cause mortality for peak O_3 exposures (95th percentile levels) in the U.S. Veterans Cohort study of approximately 50,000 male middle-aged men recruited with a diagnosis of hypertension" (p. 7-111). But the CD fails to mention that the study reported a threshold for ozone's mortality effect at 0.14 ppm for 95th percentile 1-hour ozone concentrations.[19] The 95th percentile represents the 18th worst day of the year. In other words, to exceed the ozone mortality threshold, 1-hour ozone would have to exceed 0.14 ppm more than 18 days per year. This is well above even the federal 1-hour standard, which allows only one day per year exceeding 0.125 ppm.

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[16] 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.

[17] This was based on only 3 of the 8 cities in the study that had sufficient data on all four pollutants.

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

[19] 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): 41-73.

The appropriate conclusion to draw from this study is that even in what one would expect to be a particularly sensitive subgroup of the U.S. population, the current federal 8-hour and 1-hour ozone standards both protect against premature mortality from long-term ozone exposure with a substantial margin to spare. Yet both the CD and Staff Report mislead readers to draw the opposite conclusion. Fortunately, the Staff Report draws the correct overall conclusion that there is little or no evidence for increases in mortality from chronic ozone exposure. However, this conclusion would be strengthened by an accurate summary of the Veterans Cohort results. All mentions of the Veterans study in the CD and Staff Report should be revised to include the finding of a threshold for ozone's association with premature mortality.

Health Effects of Increased Solar Ultra-Violet (UV) Exposure Due to Lower Ground-Level Ozone

Whether up in the stratosphere or near ground level, reducing ozone shielding marginally increases people's exposure to solar UV light. The CD claims these effects are too uncertain to assess:

> "Within the uncertain context of presently available information on UV-B surface fluxes, a risk assessment of UV-B-related health effects would need to factor in human habits (e.g., daily activities, recreation, dress, and skin care) in order to adequately estimate UV-B exposure levels. Little is known about the impact of variability in these human factors on individual exposure to UV radiation. Furthermore, detailed information does not exist regarding the relevant type (e.g., peak or cumulative) and time period (e.g., childhood, lifetime, or current) of exposure, wavelength dependency of biological responses, and interindividual variability in UV resistance…In conclusion, the effect of changes in surface-level O_3 concentrations on UV-induced health outcomes cannot yet be critically assessed within reasonable uncertainty" (p. 10-35).

It is strange that EPA would claim uncertainty as a reason *not* to address a potential health risk. EPA normally uses uncertainty as the justification for more stringent regulatory limits and greater safety factors. In any case, the CD's claim of uncertainty is unjustified. Back in 1997 an internal EPA analysis concluded "any decrease in atmospheric ozone (tropospheric or stratospheric) causes…an increase in the incidence of non-melanoma skin cancers…The methodology for estimating such increases (of both UV levels and skin cancer incidence) is well established."[20]

The United Nations Environment Program also has not found the effects of increased UV exposure too uncertain to estimate.[21] UNEP estimated that each 1 percent decrease in

[20] EPA never made this analysis public, but it was anonymously placed in an OMB docket and is now available at http://aei.brookings.org/admin/pdffiles/php9v.pdf.

[21] United Nations Environment Program, *Environmental Effects of Ozone Depletion: 1998 Assessment* (Nairobi, Kenya, 1998), http://www.gcrio.org/UNEP1998/UNEP98.html. The chapters of this report were also published as separate papers in the October 1998 issue of *Journal of Photochemistry and Photobiology B*, available at http://www.gcrio.org/ozone/toc.html

total atmospheric ozone results in a 1 to 2 percent increase in human exposure to UV light, and thereby to increased cancer incidence. According to UNEP, each 1 percent increase in UV exposure would likely result in 10 to 20 new skin cancers per year per million people, or 3,000 to 6,000 new cancers in a population the size of the U.S. The ozone reductions necessary to attain a national ozone standard of 0.06 or 0.07 could easily be of this magnitude or perhaps greater. The Department of Energy (1995) and Lutter (2004) have estimated similar cancer increases from lower ground-level ozone levels, as well as increases in cataracts.[22]

The harm to health from lower ozone levels is sufficient to offset much or perhaps even all of the health benefits from further ozone reductions. Of course, there are uncertainties attached to estimates of harm from increased UV exposure due to lower ground-level ozone. But these uncertainties are certainly no greater, and are arguably smaller than the large uncertainties in the epidemiological evidence of harmful effects from ozone. It is no doubt inconvenient for EPA to acknowledge and estimate the risks of marginal increases in UV exposure. But that is not an excuse for brushing off the issue. The next draft of the CD should include detailed estimates of the UV health effects due to ozone reductions, and a table comparing estimates of total health benefits to total harms.

Policy-Relevant Background (PRB) Ozone

The Executive Summaries and relevant chapters of the CD and Staff Report need to provide a more coherent summary of the sources contributing to background ozone, a more appropriate definition of the PRB, and a more scientifically defensible estimate of the PRB.

Contrary to the CD's definition, the PRB should include emissions from Canada and Mexico, as these are not directly under U.S. policy control. The PRB should also include VOC and NO emissions given off by human bodies. These are a small portion of total emissions, but might measurably add to ozone in metropolitan areas in the absence of all U.S. anthropogenic emissions. For example, based on estimates in Fenske and Paulson (1999), human VOC emissions might amount to a few tons per day in South Coast.[23] Some agricultural biogenic VOC emissions might also arguably be beyond practical policy control.

Because the ozone standard is based on extreme values, the CD should estimate a PRB based on days that are likely to have the highest PRB levels, rather basing the PRB on average ozone levels.

The CD's estimate of the PRB also seems to be based on the output of a single, low-resolution model. The CD's PRB estimates should be expanded to include high-

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[22] Department of Energy, *EPA Docket a-95-54, Iv-D-2694, Appendix B-9* (Washington, DC: March 21, 1995); R. Lutter, "Head in the Clouds Decision-Making: EPA's Air Quality Standards for Ozone," in *Painting the White House Green: Rationalizing Environmental Policy inside the Executive Office of the President*, ed. R. Lutter and J. F. Shogren (Washington, DC: Resources for the Future, 2004); R. Lutter and H. Gruenspect, "Assessing Benefits of Ground Level Ozone: What Role for Science in Setting National Ambient Air Quality Standards?" *Tulane Environmental Law Journal* 15 (2001): 85-96.

[23] J. D. Fenske and S. E. Paulson, "Human Breath Emissions of VOCs," *Journal of the Air and Waste Management Association* 49 (1999): 594-8.

resolution models and observations from areas that are relatively uninfluenced by human activities.

The next draft of the CD should include PRB estimates for a number of non-attainment areas, and with an appropriate definition of the PRB.

Ozone Chamber Studies Have Not Simulated Policy-Relevant Real-World Ozone Exposures

Comparisons of ozone exposures measured by personal monitors with ambient ozone measured at the fixed ozone monitoring sites used for regulatory compliance show that personal exposures are much lower than ambient levels, even when comparing only outdoor personal exposures to outdoor ambient levels. Thus, for example, when ambient ozone is 0.08 ppm, personal exposures outdoors are typically on the order of 40 to 60 percent lower than the ambient level.[24] And because people spend much of their time indoors, overall personal exposure is only a fraction of ambient monitor-based levels

Even people with the highest personal exposures almost always have exposure levels lower than the ambient level measured by a central ozone monitor. For example, out of about 2,100 person-days measured in a study in Alpine, California, near San Diego, there were only 3 person-days in which personal ozone exposure exceeded 0.07 ppm.[25] This is despite the fact that 27 out of the 100 days studied had ambient ozone exceeding 0.07 ppm. In probabilistic terms, for each day in which ambient ozone exceeded 0.07 ppm, an average of only 1 in 190 people (0.53 percent) was actually exposed to ozone exceeding 0.07 ppm.[26]

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[24] See, for example, R. J. Delfino, B. D. Coate, R. S. Zeiger et al., "Daily Asthma Severity in Relation to Personal Ozone Exposure and Outdoor Fungal Spores," *American Journal of Respiratory and Critical Care Medicine* 154 (1996): 633-41; A. S. Geyh, J. Xue, H. Ozkaynak et al., "The Harvard Southern California Chronic Ozone Exposure Study: Assessing Ozone Exposure of Grade-School-Age Children in Two Southern California Communities," *Environmental Health Perspectives* 108 (2000): 265-70; T. Johnson, K. Clark, K. Anderson et al., "A Pilot Study of Los Angeles Personal Ozone Exposures During Scripted Activities," Measurement of Toxic and Related Air Pollutants, Pittsburgh, Air and Waste Management Association, 1996; K. Lee, W. J. Parkhurst, J. Xue et al., "Outdoor/Indoor/Personal Ozone Exposures of Children in Nashville, Tennessee," *Journal of the Air and Waste Management Association* 54 (2004): 352-9; L. J. Liu, R. Delfino and P. Koutrakis, "Ozone Exposure Assessment in a Southern California Community," *Environmental Health Perspectives* 105 (1997): 58-65; M. S. O'Neill, M. Ramirez-Aguilar, F. Meneses-Gonzalez et al., "Ozone Exposure among Mexico City Outdoor Workers," *Journal of the Air and Waste Management Association* 53 (2003): 339-46.

[25] These ozone levels are based on 12-hour rather than 8-hour averages. The study did not report eight-hour averages. For any given 8-hour-average ozone level, the 8-hour average would of course be expected to be somewhat lower. Thus, a 12-hour average of 0.07 ppm is probably roughly the same as an 8-hour average of about 0.08 ppm.

[26] It is, of course, possible that this group of fifty people is not representative of the typical resident of Alpine. On the other hand, the results of this study are consistent with the other studies discussed in this section.

This means that a laboratory study of the effects of 0.08 ppm ozone is not representative of 0.08 ppm in ambient air, but of ambient levels of 0.12 ppm or more. When time spent indoors is taken into account, ambient levels are even higher than personal exposures.

Not only have laboratory studies used personal ozone exposures that are too high; they have used "control" exposures that are too low. Laboratory studies compare lung function when volunteers breathe, say, 0.08 ppm ozone (equivalent to at least 0.12 ppm ambient) to lung function when breathing ozone-free air. In fact, they should be using a background ozone level as the "clean air" control exposure.

Assume for the sake of argument that this background level is 0.05 ppm ambient. This would be equivalent to a personal exposure of about 0.03 ppm. If we wanted to know the potential effects of an actual ambient level of 0.085 ppm, we should then be comparing laboratory personal exposures of 0.03 ppm to represent background and 0.05 ppm to represent personal exposure for an ambient level of 0.085 ppm.

No study to date has tested the differential effects of such a small increment in ozone exposure. However, Adams (2002) tested personal exposures of 0.00, 0.04, and 0.08 ppm in heavily exercising college students.[27] There was no change in lung function between the first two exposure levels. Average lung function declined about 5 percent in going from 0.04 to 0.08 ppm, but only after 6 hours of exposure. These results suggest that laboratory tests using realistic personal exposures would find no lung-function decrements due to ozone at the level of the current federal 8-hour standard.

In other words, the evidence suggests that chamber studies with policy-relevant personal ozone exposures would not find any effects of ozone at concentrations equivalent to the current federal 8-hour standard. This is all the more striking when considering that the chamber studies are done with people performing vigorous exercise for several hours.

The CD appears to be silent on the fact that personal exposures in chamber studies are not representative of or relevant to real-world personal exposures. There is no mention of personal exposures in Chapter 6 of the CD. The CD does discuss personal exposures in the context of epidemiological studies. But here the CD creates the false impression that there is ambiguity about the difference between personal ozone exposures and ambient ozone levels.

The CD states "Some personal exposure measurements using passive samplers show O_3 exposures below those O_3 concentrations measured at outdoor stationary sites (Delfino et al., 1996; Avol et al., 1998b; Sarnat et al., 2000; Geyh et al., 2000; Brauer and Brook, 1997). However, other studies have found strong correlations between O_3 measured at stationary sites and personal monitored concentrations (Liard et al., 1999; Bauer and Brook, 1997; Linn et al., 1996; Lee et al., 2004; Avol et al., 1998b; O'Neill et al., 2003) when the time spent outdoors, age, gender, and occupation of the subjects were considered" (p. 3.54).

[27] W. C. Adams, "Comparison of Chamber and Face-Mask 6.6 Hour Exposures to Ozone on Pulmonary Function and Symptom Responses," *Inhalation Toxicology* 14 (2002): 745-64.

The fact that some studies have found "strong correlations" between stationary and personal ozone is a separate issue from whether personal ozone exposure is lower than ambient ozone levels. The CD appears to be citing these studies as evidence against personal exposures being lower than ambient levels. But this isn't what these studies show. O'Neill et al. (2003), which I cited earlier, reported that personal outdoor ozone exposures of outdoor workers in Mexico City were 60 percent lower than ambient monitor values.[28] Lee et al. (2004), also cited earlier, reported that children in Tennessee in the top 25 percent of time-spent-outdoors nevertheless experienced personal ozone exposures 80 percent lower than ambient levels.[29] Liard et al. (1999) reported that personal ozone exposure was on average lower than ozone from stationary site measurements.[30]

The initial discussion of personal exposure in the Integrative Summary also does not mention the fact that personal exposures are much lower than ambient levels (p. 8-9). The lower personal exposures are mentioned on p. 8-53, but without quantitative detail, and only in the context of epidemiological studies. Chapter 8 is silent on the implications of personal exposure vs. ambient levels for chamber studies.

The next draft of the CD should include quantitative details on the relationship between personal exposure and ambient levels, and a discussion of the fact that once the personal vs. ambient difference is accounted for, chamber studies to date suggest the current federal 8-hour standard is already protective against acute decrements in lung function.

Conclusion

In general, the 2nd Draft of the Ozone CD provides a misleading account of the evidence on ozone health effects. The CD selectively marshals evidence in support of EPA's position while mischaracterizing or omitting contrary evidence. The CD should be revised as noted above to provide a more realistic account of the weight of the evidence.

I would welcome the opportunity to discuss these comments in greater detail. Please feel free to contact me at 916.203.6309 or jschwartz@aei.org.

[28] O'Neill, Ramirez-Aguilar, Meneses-Gonzalez et al., "Ozone Exposure among Mexico City Outdoor Workers."

[29] Lee, Parkhurst, Xue et al., "Outdoor/Indoor/Personal Ozone Exposures of Children in Nashville, Tennessee."

[30] R. Liard, M. Zureik, Y. Le Moullec et al., "Use of Personal Passive Samplers for Measurement of No(2), No, and O(3) Levels in Panel Studies," *Environmental Research* 81 (1999): 339-48.

Sincerely,

/Joel Schwartz/

Joel Schwartz
Visiting Fellow
American Enterprise Institute

Appendix II

Rethinking the California Air Resources Board's Ozone Standards

American Enterprise Institute for Public Policy Research

Rethinking the California Air Resources Board's Ozone Standards

Joel Schwartz
American Enterprise Institute

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Abstract

The California Air Resources Board (CARB), California's state air-pollution regulatory agency, recently adopted the most stringent ozone air-pollution standard in the United States. Attempting to attain the new standard will impose great hardship on Californians in exchange for small and imperceptible health benefits.

By CARB's own estimates, the incremental benefits of attaining its new eight-hour ozone standard, over and above the benefits of attaining the preexisting federal eight-hour standard, include reducing the average Californian's annual risk of death by 1 in 120,000, and the risk of ending up in the hospital due to respiratory distress by 1 in 18,000. Even these small benefits are inflated, because CARB has overstated the health effects of low-level ozone exposure.

On the other hand, attempting to attain CARB's ozone standards will impose large costs on Californians, likely in the range of tens of billions of dollars per year, or a few thousand dollars per year for each California household. Californians will pay these costs in the form of higher prices, lower wages, and reduced choices, causing damage to their health, welfare, and quality of life far in excess of the tiny health improvements from additional ozone reductions.

Attaining CARB's standard is also likely to produce several hundred new cases of nonmelanoma skin cancer, a few thousand cases of cataracts, and several melanoma deaths each year by causing small increases in people's exposure to the sun's ultraviolet (UV) radiation. These harms will directly offset much of the benefit CARB predicts from attainment of its ozone standard, yet the agency did not account for or even acknowledge the increases in UV exposure in its health analysis.

CARB's actions are well-intended, but as a powerful, single-purpose agency with a staff that is passionate about air quality, it unavoidably suffers from tunnel vision—the pursuit of a single-minded goal to the point where it does more harm than good. Wealthier people lead safer and healthier lives. People made poorer by CARB's requirements will be less safe and healthy as a result.

To validate its claim to be improving Californians' health, CARB must show that attempting to attain its ozone standards will make them better off overall. This is all the more crucial because most costs of air-pollution regulations are hidden in the form of higher prices and lower wages. Thus, the people ostensibly being helped by lower ozone levels are never made aware of the real tradeoffs they've made and therefore have no way to determine whether they've struck a good bargain.

CARB's new ozone standard will cause net harm to Californians. To maximize their health and welfare, CARB should have harmonized its ozone standard with the less stringent federal eight-hour standard.

Introduction[1]

The California Air Resources Board (CARB), California's state air-pollution regulatory agency, recently adopted a stringent new eight-hour ozone standard for California and also reaffirmed the state's preexisting one-hour standard.[2]

Both of California's ozone standards are more stringent than the Environmental Protection Agency's eight-hour standard, which is the one all states are required to attain under the federal Clean Air Act. California's one-hour standard is slightly more stringent than the federal eight-hour standard, while its new eight-hour standard is by far the toughest ever adopted in the United States. Areas of the state that exceed the federal eight-hour ozone standard exceed the new California eight-hour standard two to three times as often. While many regions of California are in or near full compliance with the federal standard, virtually the entire state violates CARB's new eight-hour standard.

CARB's staff is dedicated to delivering clean air to all Californians. But attempting to attain CARB's ozone standards will impose great hardship on Californians in exchange for small and imperceptible health benefits.

By CARB's own estimates, the incremental benefits of attaining its one-hour and eight-hour ozone standards, over and above the benefits of attaining the preexisting federal eight-hour standard, include reducing deaths by 0.13 percent, respiratory hospital admissions by 0.55 percent, and emergency room (ER) visits for childhood asthma by 0.84 percent. According to CARB, these reductions will amount each year to 300 fewer deaths, 2,000 fewer respiratory hospital admissions, and 314 fewer ER visits due to childhood asthma. In terms of risk reduction, the average Californian's annual risk of death will decline by 1 in 120,000, the risk of ending up in the hospital due to respiratory distress will drop by 1 in 18,000, and the risk to child asthmatics of going to the ER for asthma will drop by 1 in 4,000. These benefits are small, and even they are inflated, because CARB overstated the harm from low-level ozone exposure.

On the other hand, attempting to attain CARB's ozone standards will impose large costs on Californians. The most recent estimates indicate it will cost $4 billion per year for just the Los Angeles metropolitan region to attain the federal one-hour ozone standard, and an additional $17 billion per year to attain the federal eight-hour standard. Statewide costs will be even greater.

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[1] This paper is a revised version of comments submitted in March 2005 to the California Air Resources Board when it was considering adoption of its new ozone standards.

[2] California Air Resources Board, "California Adopts New Ozone Standard; Children's Health Focus of New Requirement," April 28, 2005. For additional information on CARB's structure, mission, and authority, see http://www.arb.ca.gov/html/brochure/arb.htm.

Attaining CARB's new eight-hour standard will cost additional tens of billions per year. Californians will pay these costs in the form of higher prices, lower wages, and reduced choices, causing damage to their health, welfare, and quality of life far in excess of the tiny health improvements from additional ozone reductions. For example, if the incremental cost of attaining CARB's eight-hour standard is $20 billion per year—which is likely to be a substantial underestimate of the real cost—this would amount to an average of about $1,700 per California household. It is likely that CARB's eight-hour standard is set below background peak ozone levels in some areas of the state, including parts of the San Bernardino, Riverside, Fresno, and Bakersfield areas. If so, then the standard is unattainable in much of the state, regardless of cost.

Risk analysts estimate that each $17 million in additional regulatory costs induces one additional statistical death by diverting resources away from other risk-reduction expenditures, such as for safer cars or additional health care. Based on this finding, and assuming CARB is correct in its claim that attaining its standard will avoid three hundred premature deaths each year, its eight-hour standard will kill more people than it saves if the incremental cost of attaining the standard is greater than about $5 billion per year. Attempting to attain the standard will actually cost several times more than this amount, and the new standard is therefore likely to kill hundreds more people per year than it saves. If low-level ozone exposure does not increase mortality, as suggested by the weight of the evidence, then CARB's standard will do more harm than good even if attaining it costs substantially less than $5 billion per year.

Attaining CARB's standards is also likely to cause several hundred new cases of nonmelanoma skin cancer, a few thousand cases of cataracts, and several melanoma deaths each year by causing small increases in people's exposure to the sun's ultraviolet (UV) radiation. These harms will directly offset much of the health benefit CARB predicts from attainment of the standards, yet the agency did not account for or even acknowledge the increases in UV exposure in its health analysis.

CARB has not attempted to estimate what Californians will have to pay to attain its ozone standards. In fact, CARB claims in its staff report on the new ozone standard that its air pollution standards do not impose any costs on Californians, because "standards simply define clean air."[3] According to CARB, the full effects of trying to attain its ozone standards will be weighed when it comes time to adopt the regulations necessary for attainment.

The agency did provide a detailed estimate of the predicted health benefits of attaining its standards. But by CARB's logic, just as it imposes no costs, a standard confers no benefits. Only the act of attempting to attain a pollution standard can impose costs or confer benefits. By omitting discussion of costs while discussing benefits in detail, CARB has created the false impression that the predicted health benefits of its

[3] 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, p. 1-6.

ozone standards can somehow be delivered without imposing any offsetting hardships on the people who ostensibly would be helped by the tougher standards.

California law does not provide CARB with the authority to mandate attainment of its ozone standards. However, the standards should not be seen as merely symbolic. CARB has broad regulatory authority over many sources of air pollution, including motor vehicles, and can invoke this authority in the service of attaining its stricter standards. Equally important, CARB's new eight-hour standard has become the government-sanctioned delineation between "safe" and "unsafe" air. Because the new standard is more stringent, it will be exceeded much more frequently than the old one, creating more ozone-alert days and bigger, scarier numbers of "bad-air" days in regulators' and activists' reports and press releases. The result will be greater public fear for any given level of ozone, and greater pressure for bold action to alleviate the alleged crisis. CARB's ozone standards will thus become their own justification, without CARB ever having to provide evidence that attempting to attain the standards will provide *net* benefits to the people of California.

CARB's actions are well-intentioned, but as a powerful, single-purpose agency with a staff that is passionate about air quality, the agency unavoidably suffers from tunnel vision—the pursuit of a single-minded goal to the point where it does more harm than good. Wealthier people lead safer and healthier lives. People made poorer by CARB's requirements will be less safe and healthy as a result.

To validate its claim to be improving Californians' health, CARB must show that attempting to attain its ozone standards will make people better off overall. This is all the more crucial because most costs of air-pollution regulations are hidden in the form of higher prices, lower wages, and reduced choices. Thus, the people ostensibly being helped by lower ozone levels are never made aware of the real tradeoffs they've made, and therefore have no way to determine whether they've struck a good bargain.

When the Environmental Protection Agency adopted the federal eight-hour ozone standard in 1997, it predicted that the social benefits of attaining the standard would be only one-half the social costs. In other words, even the EPA concluded that attempting to attain its own eight-hour standard would make Americans worse off overall. Moreover, outside analysts showed that the real costs of attaining the standard would likely exceed the EPA's prediction by more than a factor of ten, making the net effects of the standard far worse than even the EPA's own gloomy prediction would suggest.

CARB's eight-hour ozone standard will cause far more net harm than the federal standard, because the incremental costs of attaining CARB's standard will be far larger, while the incremental benefits will, at best, be about the same. To maximize Californians' health and welfare, CARB should have adopted state ozone standards equivalent in stringency to the federal eight-hour standard.

Comparing Federal and California Ozone Standards

Ozone standards have three major components: an averaging time; a maximum ozone concentration level, usually reported in parts per million (ppm) or parts per billion (ppb); and a maximum number of days that the ozone level can be exceeded before the standard is violated.[4] The averaging time is the number of hours each day over which the ozone level is averaged. For example, one-hour standards are based on the single hour each day with the highest ozone reading; eight-hour standards are based on the eight-hour period each day with the highest average ozone reading.

The stringency of a standard depends on a combination of the maximum ozone level and the number of exceedance days allowed. Averaging time has no systematic effect on the stringency of the standard. For any given averaging time, a level and exceedance limit can be set that make a given standard roughly equivalent to a standard based on any other averaging time.[5]

The federal eight-hour ozone standard is 0.085 ppm. A monitoring location violates the standard if the average of the fourth-highest daily ozone reading from each of the last three years reaches 0.085 ppm. In practice, this means that violating the standard requires an area to average at least four or five days per year in which eight-hour-average ozone levels reach at least 0.085 ppm. The federal one-hour standard is set at 0.125 ppm, with up to three exceedance days allowed in any consecutive three-year period.

CARB's one-hour standard is set at 0.095 ppm, and its new eight-hour standard is set at 0.070 ppm. In both cases, no exceedances are allowed. An entire region, say, the Los Angeles metropolitan area or the San Francisco Bay Area, falls into "nonattainment" of a given standard if it has at least one monitoring location that violates the standard.

Setting the eight-hour standard at 0.095 ppm would have made it roughly equivalent to the current federal one-hour standard in stringency. On the other hand, a one-hour standard set at about 0.10 ppm would be about as stringent as the current federal eight-hour standard. California's standards are more stringent than the federal standards both because California's standards are exceeded at a lower ozone level, and because no exceedances are allowed before the standard is violated. Table 1 summarizes the relationships among the various federal and California ozone standards.

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[4] EPA's eight-hour ozone standard is not based on a number of exceedances, but on the average of the fourth-highest daily peak eight-hour ozone reading from each of the last three years. In practice, this is roughly equivalent to a maximum allowable exceedance rate of four to five days per year.

[5] I say "roughly," because the ratio between, say, one-hour and eight-hour ozone levels varies somewhat from region to region. Thus, one can always create a standard that will have the same stringency on average, but changing the averaging time can change somewhat the relative stringency of a standard among several different regions.

Compared with the federal eight-hour ozone standard, CARB's eight-hour standard will greatly increase the number of exceedance days per year at California's ozone monitoring sites. Figure 1 compares exceedances per year in each California "air basin" under the current federal standard with the incremental increase in exceedances for CARB's standard. The left-hand graph displays data for the worst monitoring location in each region, while the right-hand graph displays data for the average location in each region. CARB's standard will increase the number of exceedances per year by about a factor of 1.7 in the worst location in South Coast (Crestline) and by a factor of 2.0 or 3.0 in most areas of the state.

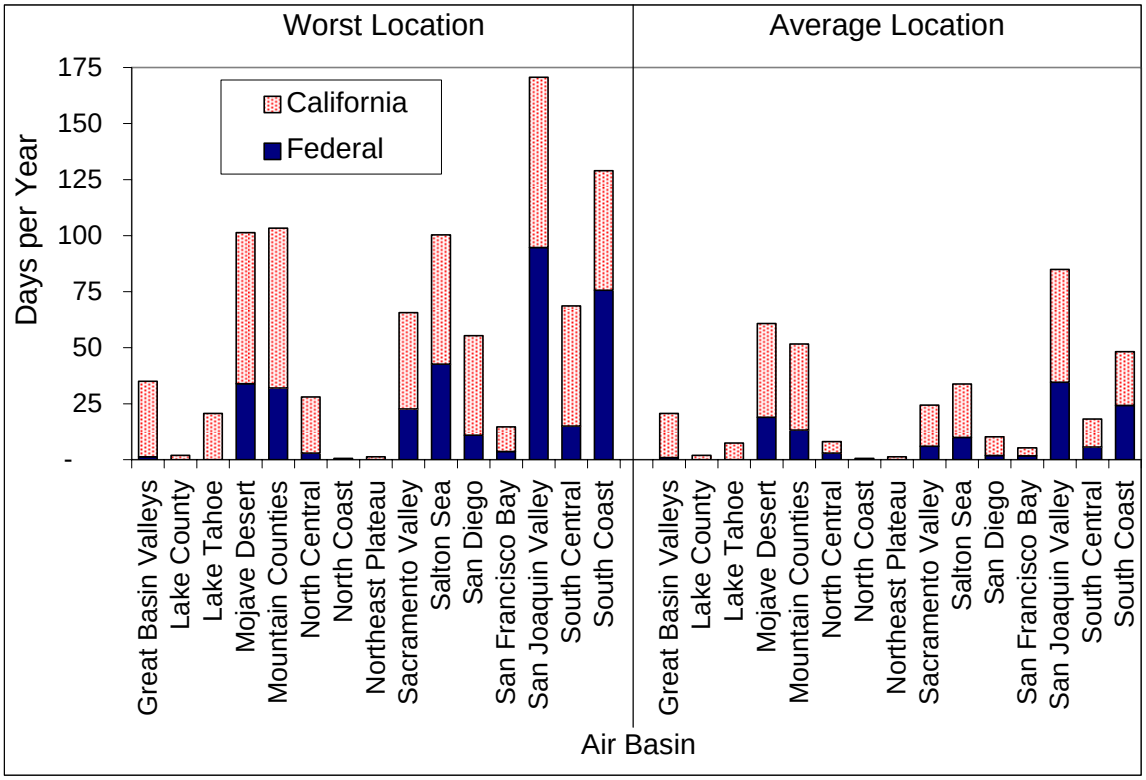
Figure 2 compares exceedance rates for all four standards in five of California's most populous air basins. The graph gives the average annual number of exceedances at the worst site in each air basin during 2001–03 for each of the four standards. Note that the current CARB one-hour standard is somewhat more stringent than the current federal eight-hour standard, but not nearly as stringent as CARB's eight-hour ozone standard.

Based on data for 2001–03, figure 3 compares the highest eight-hour ozone reading at the worst site in each of California's air basins to the levels of the federal and California eight-hour ozone standards (dashed and dotted lines, respectively). However, as explained in the notes to figure 3, because the federal eight-hour standard is based on the fourth-highest reading each year, the highest eight-hour ozone reading in a given year will be somewhat higher than 0.085 ppm for areas that comply with the standard.

Table 1. Comparison of Federal and California Ozone Standards

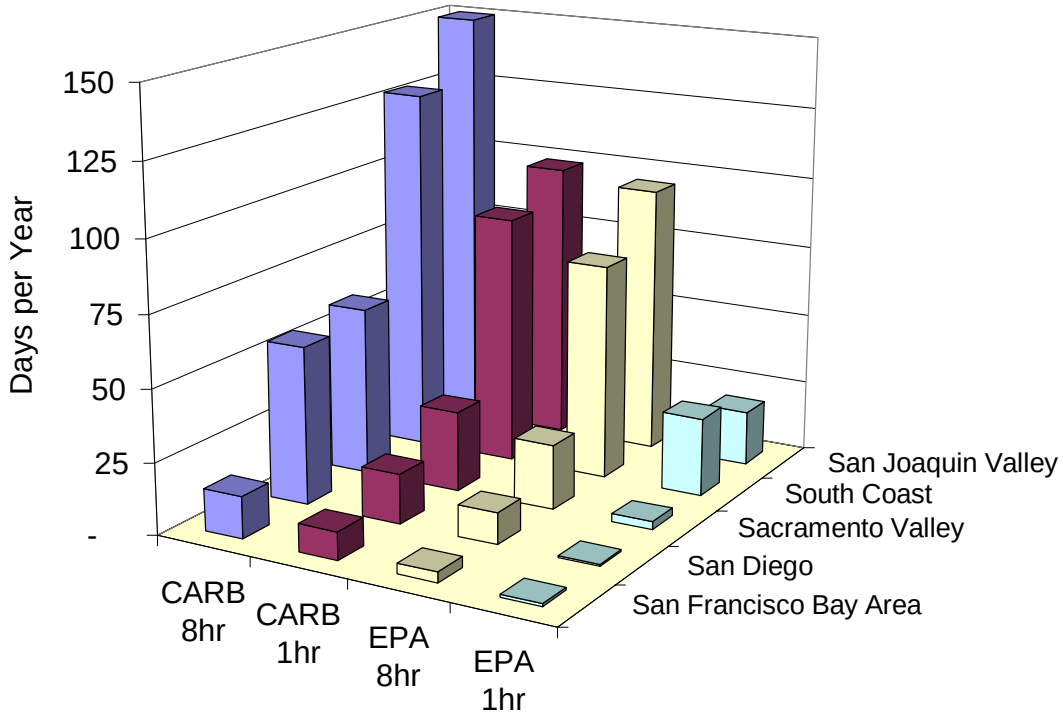
Averaging Time	One-Hour		Eight-Hour	
	Federal	California	Federal	California
Ozone Level (ppm)	0.125	0.095	0.085	0.070
Exceedances Allowed	No more than three in any consecutive three-year period	None	Average of fourth-highest reading in each of last three years must be less than 0.085 ppm, equivalent to an average of no more than about four to five exceedances per year	None
Notes	Substantially less stringent than federal eight-hour	Slightly more stringent than federal eight-hour		Substantially more stringent than federal eight-hour

Figure 1. Number of Eight-Hour Ozone Exceedance Days per Year at the Worst and Average Locations in Each California Air Basin; Federal Eight-Hour Standard and Incremental Effect of CARB's Eight-Hour Standard; Data for 2001–03



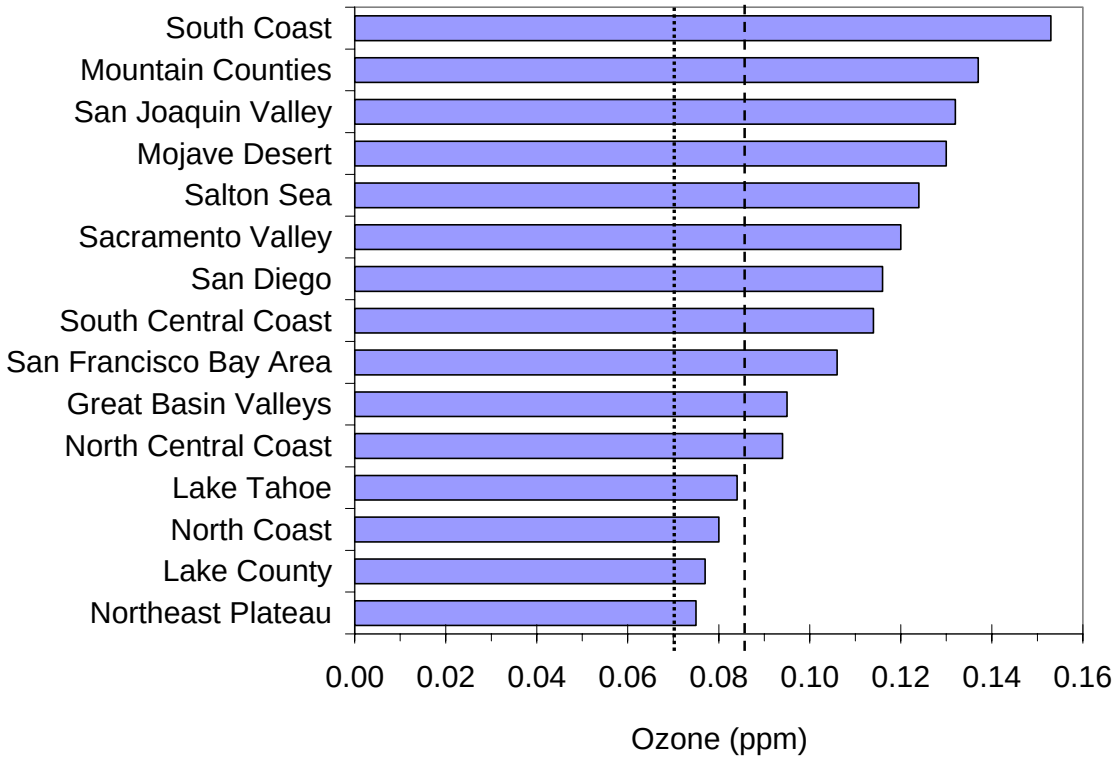
Notes: "South Coast" is the Los Angeles metropolitan area.
Source: California Air Resources Board, *California Ambient Air Quality Data 1980-2003*, data CD-ROM #PTSD-05-020-CD, January 2005.

Figure 2. Average Number of Exceedance Days per Year at Worst Location in Each Air Basin, Based on Four Different Ozone Standards, 2001–03



Notes: "South Coast" is the Los Angeles metropolitan area.
Source: California Air Resources Board, *California Ambient Air Quality Data 1980-2003*.

Figure 3. Highest Eight-Hour Ozone Reading During 2001–03 in Each of California's Air Basins



Notes: "South Coast" is the Los Angeles metropolitan area. The dashed line marks the federal eight-hour ozone standard, while the dotted line marks CARB's California standard. However, note that the federal standard is based on the fourth-highest daily ozone reading each year, rather than the highest reading. The result is that the federal eight-hour standard allows peak ozone readings to be somewhat higher than the 0.085 ppm standard. For example, looking at the five regions from figure 2 and taking the year with the highest peak eight-hour ozone during 2001–03 in each of those regions, the ratio of the highest day to the fourth-highest day ranged from 1.15 to 1.29. Thus, after attaining the federal eight-hour standard, eight-hour ozone on the worst day of the year could have been as high as 0.098 ppm to 0.110 ppm in these areas.

Source: California Air Resources Board, *California Ambient Air Quality Data 1980-2003*.

CARB's Estimate of Health Benefits

Based on CARB's estimates, reducing ozone from attainment of the current federal eight-hour standard to attainment of CARB's California standards would delay 0.13 percent of all deaths, and avoid 0.55 percent of all respiratory hospital admissions and 0.84 percent of all asthma-related ER visits.[6] In other words, based on CARB's estimates, the incremental benefit of attaining its California ozone standards, would be to delay about 1 in 800 deaths, and to avoid 1 in 180 respiratory hospital admissions and 1 in 120 asthma ER visits. CARB estimates that going from current ozone levels down to attainment of the federal eight-hour standard would result in similar health benefits as going from the federal eight-hour standard down to CARB's eight-hour standard. These results suggest that even at current levels, ozone is having a small effect on overall public health.

CARB does, however, estimate that reducing ozone from current levels down to its new standard will produce a large decline in school absences, reducing them by nearly nine percent. This would increase attendance by an average of about one-half day per year per student. Since only about half of school absences are due to respiratory illness, this means a reduction in respiratory-related absences of nearly 18 percent.

Figure 4 shows, based on CARB's estimates, the percentage of each ozone-related health effect that would be avoided by going from eight-hour ozone levels during 2001–03 down to the current federal eight-hour ozone standard, and the additional incremental benefit of attaining CARB's eight-hour standard. The incremental benefit of CARB's standard accounts for 48 percent of the total benefits. Table 2 displays the values plotted in the graph.

The inset graph in figure 4 has a shortened vertical scale to allow easier identification of the incremental benefits of CARB's standard. The results are derived from CARB's estimates of the baseline rate of each health effect in California and the number of cases avoided by attaining CARB's standard. The methodology is explained in more detail in the footnote.[7]

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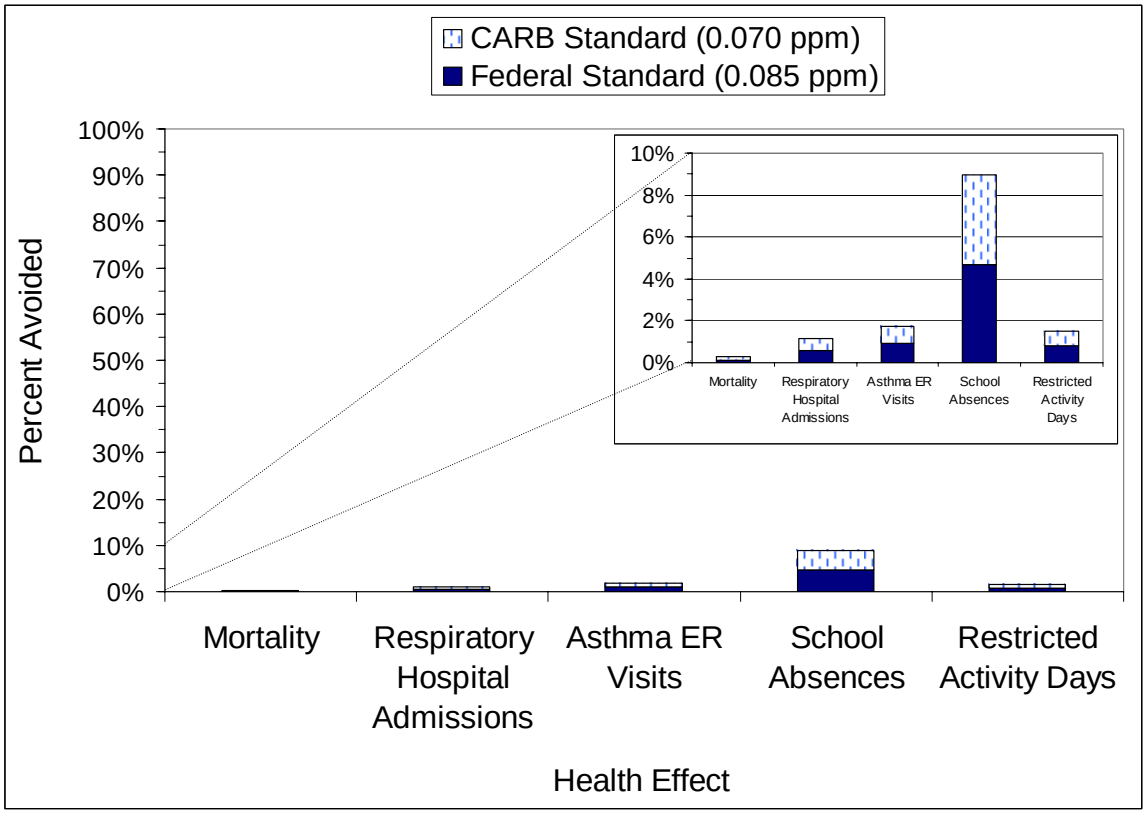
[6] Death can, of course, only be delayed, rather than avoided altogether. Thus, reducing pollution should be seen as potentially avoiding *premature death*. The more precise benefit question then becomes how many years of life are saved by reducing ozone. This question depends, for each premature death avoided, on whether reducing ozone typically extends life by a few days or weeks, or by several years. I return to this question later in this analysis.

[7] To calculate the percent reduction in a given health effect (for example, asthma ER visits) attributable to ozone reductions, I used CARB's estimate of cases avoided for each health effect due to statewide attainment of the new standard (table B-5, p. B-25 in CARB's staff report) and divided by the total number of cases. To calculate the total number of cases, I used CARB's county-by-county estimates of baseline incidence rates for each condition (p. B-52) and combined them with the 2003 population of each county (http://www.dof.ca.gov/HTML/DEMOGRAP/E-2_Jul04.xls) to calculate total incidence statewide. In the case of childhood asthma, the population was limited to children up to seventeen years of age, and an assumed asthma prevalence of 13 percent. For school absences, the population was limited to children five to seventeen years of age. In the case of restricted activity days, the population was limited to people

Figure 4. Percent of Harmful Health Effects Avoided by Going from Current Air Quality to the Federal Eight-Hour Ozone Standard and to CARB's Eight-Hour Standard



Notes: The dark portion of each bar gives the predicted benefits of attaining the federal eight-hour ozone standard. The light portion gives the predicted additional incremental benefits of attaining CARB's eight-hour standard.

Sources: CARB's staff report estimates of the total rate of each condition by county in California and the number of cases avoided by attaining CARB's standard (see appendix B). CARB's staff estimated the incremental benefits of its standard over and above the federal eight-hour standard in a presentation to CARB's governing board at the adoption hearing for the standard, available at http://www.arb.ca.gov/research/aaqs/ozone-rs/4-28-05pres.pdf. California Department of Finance county population estimates for 2003 are at http://www.dof.ca.gov/HTML/DEMOGRAP/E-2_Jul04.xls.

eighteen years of age and older. CARB estimates that its new eight-hour standard accounts for 48 percent of the total benefits of going from ozone levels during 2001–03 to full statewide attainment of CARB's standard.

Table 2. Percent of Health Effects Avoided by Going from Current Air Quality to the Federal Eight-Hour Ozone Standard and to CARB's Eight-Hour Standard

Health Effect	Attainment of Federal Eight-Hour Standard (0.085 ppm)	Incremental Benefits of California Eight-Hour Standard (0.070 ppm)	Total Reduction in Cases
Mortality	0.14%	0.13%	0.27%
Respiratory Hospital Admissions	0.60%	0.55%	1.15%
Asthma ER Visits	0.92%	0.84%	1.76%
School Absences	4.69%	4.26%	8.95%
Restricted Activity Days	0.80%	0.72%	1.52%

CARB estimated benefits for only a subset of potential short-term ozone health effects and did not estimate reductions in any potential long-term effects, citing in both cases uncertainty or insufficient data for quantitative estimates. I will show later that even current California ozone levels are unlikely to be causing long-term health effects. The case for long-term health effects at ozone levels below the current federal eight-hour standard—that is, within the range of ozone levels addressed by CARB's standard—is weaker still.

Activists, regulators, and journalists generally portray air pollution as a major factor in people's health, giving the impression that most respiratory and cardiovascular distress is due to poor air quality.[8] But by CARB's own estimates, even reducing ozone down to the *current* federal eight-hour ozone standard would mitigate only a small fraction of death, disease, and discomfort. The potential health improvements from attaining CARB's standard are similarly small. Yet even here, public comments by environmental groups on the standard create the impression that people will notice large improvements in their health due to ozone reductions.[9]

The EPA's estimates of the benefits of ozone reductions are somewhat smaller than CARB's. The EPA estimates that national attainment of the federal eight-hour ozone standard would reduce various health effects by between 0.02 percent (asthma hospitalizations) and 0.11 percent (school absences).[10] Despite the fact that CARB's own

[8] See, for example, B. Anderson, "Fresno Is Second for Smog," *Fresno Bee*, May 1, 2003; Earthjustice, *Urgent Cases: Valley Air: Agricultural Exemptions (Title V)*, 2001, http://www.earthjustice.org/urgent/display.html?ID=65; M. McCabe, "The Foul Air in the Smog-Choked San Joaquin Valley Is Blamed for an Epidemic of Respiratory Problems That Leave Residents Breathless," *San Francisco Chronicle*, August 17, 2002.

[9] See, for example, American Lung Association, Environmental Defense, Sierra Club et al., *Support Proposed Revisions to California's Ambient Air Quality Standards for Ozone*, September 1, 2004, http://www.arb.ca.gov/research/aaqs/ozone-rs/comments/ala.pdf.

[10] B. J. Hubbell, A. Hallberg, D. R. McCubbin, et al., "Health-Related Benefits of Attaining the 8-Hr Ozone Standard," *Environmental Health Perspectives* 113 (2005): 73–82. I calculated the percentages based on

estimates imply ozone is having only a tiny effect on public health, its staff report misleadingly implies it is causing great harm, and that reducing it will have large health benefits. For example, the executive summary states that "the statewide potential for significant health impacts associated with ozone exposure is large and wide-ranging."[11]

CARB's estimate of the health benefits of lower ozone presented above, though small, is likely to be a best-case estimate. Later in this paper I will show that the true health benefits of attaining the standard are likely to be substantially lower. CARB's estimates for reductions in premature mortality and school absences are particularly implausible.

The fact that ozone accounts for only a tiny fraction of the burden of disease does not by itself vitiate the case for a tougher standard. Based on CARB's estimates, the incremental benefit of CARB's standard would be, for example, to delay three hundred deaths and avoid two thousand hospital admissions each year. Without a doubt, we would all choose to save lives and stop hospital visits if we could. But couching the question in this way suggests that the only choice we face is between saving three hundred lives per year or not saving them; or between stopping two thousand hospital admissions per year or not stopping them.

The real choice we face is more complicated. First, ozone reductions are costly, and these costs are ultimately paid by consumers, crowding out expenditures for other needs and desires that affect health, welfare, and quality of life. I show below that these costs are far larger—likely in the range of tens of billion of dollars per year, or a few thousand dollars per household—than the small benefits of ozone reductions. As a result, the measures necessary to attain CARB's standard will cause net harm to Californians' health and welfare in a number of ways:

- Ozone, whether up in the stratosphere or near ground level, blocks the sun's ultraviolet light. Thus, reducing ozone will also increase people's exposure to solar UV light, increasing the risk of developing skin cancer and cataracts.

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the EPA's estimates of the overall underlying rate of each health condition and of the number of cases of each condition that would be avoided by attaining the federal eight-hour ozone standard. The EPA's benefit estimates are somewhat smaller than CARB's due to two factors: first, ozone levels are already relatively low in most of the country, when compared with some of California's populous areas, so there is less ozone to reduce and therefore fewer benefits to be had; and second, the EPA assumed ozone would be reduced only to the eight-hour standard in areas that violate the standard and not at all in areas that already attain the standard. However, ozone nonattainment in a given area is based on the monitoring location with the worst ozone level. Measures necessary to reduce ozone at this peak location would also reduce ozone in other areas of a nonattainment region, presumably including areas that already attain the standard. CARB assumed ozone would be reduced in all areas of California, regardless of attainment status (so long as ozone is above an assumed background level of 0.04 ppm), as a side effect of measures implemented to meet the standard at the peak site in a given air basin. CARB estimates that only about 20 percent of the total benefits it predicts for ozone reductions are due to reducing ozone down to the standard. The other 80 percent of the benefits come from reducing ozone below the standard.

[11] California Air Resources Board, *Review of the California Ambient Air Quality Standard for Ozone*, p. 1-2.

The harm from lower ozone levels would directly offset much of the benefit CARB claims for lower ozone levels.

- Even if reducing ozone provides *net* improvements in people's health, it is by a large margin one of the most expensive risk-reduction options available per year of life saved. Investing in ozone reductions will result in more death and disease than if the same funds are devoted to other measures that save more lives per dollar invested.

- Much of the health benefit CARB claims for ozone reductions is based on a selective and flawed reading of the evidence and is unlikely actually to materialize.

- About 80 percent of the benefits CARB claims for ozone reductions are due to reducing ozone from levels that already comply with its standard. Since CARB claims its standard protects public health with an adequate margin of safety, no benefits should be claimed for reducing ozone below the level of the standard.

I discuss each of these issues in detail below.

The Health Benefits of Attaining CARB's Standard Are Lower than CARB Projects

Claims about the health benefits of reducing ozone below the relatively low levels experienced today are based mainly on epidemiological studies reporting small statistical associations between ozone and health outcomes. But a number of researchers have argued that many of these statistical associations are likely artifacts of the statistical methods themselves and of publication bias—that is, the tendency of researchers and journal editors to publish only studies or analyses that find an association between pollution and health, and not ones that find no association—rather than being indicative of a real cause and effect relationship between low-level pollution and health.

In other words, not only are we faced with tradeoffs between the costs and benefits of ozone reduction, and between the relative benefits of reducing ozone versus doing something else to improve health; we also run the risk that the hoped-for benefits of ozone reductions will not materialize. Thus, when faced with a choice among a number of different health-improvement measures, we also have to consider the relative likelihood that the hoped-for health benefits of ozone reductions will actually materialize.

Ozone and Risk of Death

Death is by far the most severe harm CARB attributes to ozone. Based on CARB's estimates, attaining the federal eight-hour ozone standard would delay 330 deaths each year, with an additional 300 deaths delayed by attaining CARB's ozone standards. These estimates are based on the results of epidemiological studies that reported statistical associations between daily ozone levels and nonaccidental deaths. Based on a number of studies in the literature, CARB assumes that each 0.010 ppm decrease in twenty-four-hour-average ozone levels (0.025 ppm for one-hour ozone and 0.019 for eight-hour ozone, based on CARB's assumed conversion factors) is associated with a 1 percent decrease in daily deaths.[12] For reasons described below, this presumed relationship

[12] California Air Resources Board, *Review of the California Ambient Air Quality Standard for Ozone*, p. B-11. CARB uses a conversion factor of 1.33 to go from eight-hour to one-hour ozone levels. This is too high. Inspection of actual daily eight-hour and one-hour ozone data shows that the average ratio in California's major air basins ranges from 1.14 (San Joaquin Valley) to 1.22 (South Coast). Using these more realistic ratios could either increase or decrease CARB's predicted mortality (and other) benefits for eight-hour ozone reductions, depending on whether the conversion was from one-hour to eight-hour or from eight-hour to one-hour. This depends in turn on whether the particular studies CARB used for a given health effect were based on one-hour or eight-hour ozone levels. In either case, the under- or overestimate would be on the order of 10–15 percent.

My estimate of one-hour/eight-hour ratios includes all site-days during 2000–03 for which daily peak eight-hour ozone exceeded 0.04 ppm. The ratio for days with eight-hour ozone greater than 0.07 ppm was similar to that for days with ozone less than 0.07 ppm (for example, 1.23 versus 1.21 for South Coast).

In some cases, health-effects studies were based on twenty-four-hour-average ozone levels. CARB assumed that one-hour peak ozone levels are 2.5 times greater than twenty-four-hour ozone levels. I was not able to check this conversion factor as part of this study. However, any errors in the conversion factor would likewise introduce errors into CARB's assumed dose-response functions for ozone health effects. Given that the ratio of one-hour to eight-hour ozone levels is less than CARB assumes, it is likely that one-

between ozone and daily deaths is likely to be biased high as a result of data-mining and publication bias.

Data-Mining. Data-mining refers to the huge number of possible mathematical models that relate air pollution to health effects. Because the true biological relationship between various air pollutants and health is unknown, and because other factors that are correlated with air pollution (such as weather) also affect health, if one tries out enough different combinations of variables there is a good chance that at least a few will give statistically significant associations between air pollution and health. However, these results could be due to random chance rather than real cause and effect relationships.

In a clinical study of a drug, people are assigned randomly to treatment and control groups. Any differences between the two groups can then confidently be assumed to be due to the drug, because random assignment should eliminate all other systematic differences between the two groups. Randomization is not ethical or practical in real-world studies of air pollution. Instead, researchers observe daily air-pollution levels and nonaccidental death rates in a given area and look for statistical associations between the two using a mathematical technique called regression analysis.

The problem is that any of a number of factors could be the real cause of a given death on a given day. If any of these factors is correlated with ozone levels, researchers can attribute deaths to ozone when they are actually caused by some other factor. As a recent review of air-pollution epidemiology studies concluded,

> Estimation of very weak associations in the presence of measurement error and strong confounding is inherently challenging. In this situation, prudent epidemiologists should recognize that residual bias can dominate their results. Because the possible mechanisms of action and their latencies are uncertain, the biologically correct models are unknown. This model selection problem is exacerbated by the common practice of screening multiple analyses and then selectively reporting only a few important results.[13]

To mitigate some of the problems inherent to using observational data rather than randomized, controlled studies to assess the health effects of air pollution, a few researchers have used a technique known as Bayesian model averaging. Though mathematically complicated, the technique is simple in principle: Take all possible

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hour/twenty-four-hour and eight-hour/twenty-four-hour ratios are also less than CARB assumes, since twenty-four-hour ozone includes eight-hour ozone, and variation in the daily eight-hour peak is the main driver of daily variation in twenty-four-hour ozone levels. If this reasoning is correct, then, all else equal, CARB would underestimate health effects of ozone in cases where it used studies based on twenty-four-hour ozone to predict health effects of one-hour and eight-hour ozone variations, and overestimate health effects of ozone in going from one-hour or eight-hour ozone to twenty-four-hour ozone.

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

regression models relating air pollution and other factors, such as weather, to health outcomes; weight the models according to how well they fit the actual data; then take a weighted average of the results. This gives an average and an uncertainty range for the correlation between, say, ozone and death, after controlling for the effects of other factors that could affect health.

Factors that should be accounted for in a study of the relationship between air pollution and health include

- various air pollutants, such as ozone, particulates, and carbon monoxide;
- weather conditions, such as temperature, humidity, wind speed, and barometric pressure;
- timing of effects. Current-day pollution or weather might be a culprit, for instance, but delayed effects from the previous few days might be important as well;
- interactions between variables. For example, ozone might cause death only on very hot days, or only in the presence of another pollutant; and
- long-term trends in mortality that are unrelated to air pollution, such as flu epidemics or other mortality trends related to season, or longer-term mortality trends related to changing health habits such as diet, exercise, or smoking.

The result is literally hundreds of potential explanatory variables and trillions of potential models. A recent study that assessed the relationship between ozone and mortality using Bayesian model averaging concluded that the effect of ozone on mortality is statistically indistinguishable from zero.[14] According to the researchers, "Models that elicit statements of the form 'ozone has no effect on mortality' receive the most support from the data," and "a method that presents results from a single regression may lead researchers to make misleading inferences about pollution–mortality effects, thereby seriously underestimating the true uncertainty in the statistical evidence."

CARB's estimate of the mortality benefits of reducing ozone is based on just such point estimates from single regressions, and therefore it overestimates the certainty of the evidence. Once model uncertainty is included in the estimate, the most plausible conclusion is that ozone has no effect on mortality.

Publication Bias. Publication bias refers to the tendency of researchers to seek publication of, and for journals mainly to accept, those studies that find a statistically

[14] 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): 30–54.

significant effect, while not publishing studies that don't find an effect.[15] Even without accounting for model uncertainty, accounting for publication bias significantly reduces the estimated relationship between ozone and mortality. For example, in its draft staff report, CARB based its mortality benefit estimates mainly on the results of a World Health Organization (WHO) meta-analysis of the ozone–mortality relationship.[16] The WHO analysis combined the results of several single-city studies to estimate an overall relationship between ozone levels and mortality. Such meta-analyses are subject to publication bias, on account of the possibility that other studies have been performed but were not published because they did not identify an effect. Since these studies are not known or available, they cannot be included in a meta-analysis, resulting in overestimation of a given health risk.

The WHO meta-analysis estimated that its ozone–mortality relationship should be adjusted downward by one-third due to inferred publication bias. The ozone–mortality relationship was borderline statistically insignificant after this adjustment. The WHO report includes the following cautionary discussion about the problem of publication bias:

> 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.[17]

Another major study, the National Morbidity, Mortality, and Air Pollution Study (NMMAPS), does not suffer from publication bias, because it applies the same analytical methods to pollution and mortality data for ninety-five different U.S. cities. A recent NMMAPS report on the relationship between ozone and mortality reported an ozone effect 70 percent lower than the result derived from meta-analysis of single-city studies,

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[15] Publication bias is a well-documented problem in a range of disciplines. See, for example, V. M. Montori, M. Smieja and G. H. Guyatt, "Publication Bias: A Brief Review for Clinicians," *Mayo Clinic Proceedings* 75 (2000): 1284–8; A. Thornton and P. Lee, "Publication Bias in Meta-Analysis: Its Causes and Consequences," *Journal of Clinical Epidemiology* 53 (2000): 207–16.

[16] 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. A meta-analysis combines results of several different studies of a given health effect to derive an overall estimate of the size of the given effect.

[17] Ibid.

and concluded that publication bias inflates the ozone health effects estimated via meta-analyses.[18]

Both the WHO analysis and NMMAPS reported additional results that add to concerns about whether ozone is causing mortality increases. For example, the WHO analysis reported no association between ozone and respiratory mortality, while the association of ozone with cardiovascular mortality was the same as for all-cause, nonaccidental mortality. These results are biologically implausible. If ozone exerts its effects through the respiratory system, one would expect a *greater* ozone effect on respiratory and cardiovascular mortality than on all-cause mortality. Furthermore, after adjusting for publication bias, the WHO analysis concluded that higher ozone was associated with *lower* respiratory mortality. In NMMAPS, higher ozone levels were likewise associated with lower mortality in about one-third of the cities in the study.[19]

CARB Fails to Account for Data-Mining and Publication Bias. CARB fails to account for these factors in its estimates of mortality benefits from ozone reductions. For example, CARB's "central estimate" of a 1 percent increase in premature mortality per 0.019 ppm increase in eight-hour ozone is essentially based on meta-analyses that are uncorrected for publication bias. CARB's low-end estimate is an 0.5 percent increase in mortality. But this "low-end" is more like a publication-bias-corrected "central estimate." Given that NMMAPS, like several other studies, reported lower mortality with higher ozone for many cities, and accounting for model uncertainty, a defensible low-end estimate of ozone's mortality effect should be no higher than zero. Indeed, the evidence is consistent with a central estimate of zero.

CARB recently updated its ozone staff report to include the results of three new meta-analyses of daily ozone levels and mortality published in the July issue of the journal *Epidemiology*.[20] Each meta-analysis was performed by a different research group, but all three were commissioned by the EPA. The range of results from the three studies is such that each 0.010 ppm increase in daily one-hour ozone was associated with a 0.35–0.41 percent increase in daily mortality. To put this on the same terms as CARB's results

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[18] M. Bell, J. Samet and F. Dominici, *Ozone and Mortality: A Meta-Analysis of Time-Series Studies and Comparison to a Multi-City Study (the National Morbidity, Mortality, and Air Pollution Study)* (Baltimore: Johns Hopkins School of Public Health, July 19, 2004), http://www.bepress.com/cgi/viewcontent.cgi?article=1057&context=jhubiostat.

[19] M. L. Bell, A. McDermott, S. L. Zeger, et al., "Ozone and Short-Term Mortality in 95 US Urban Communities, 1987–2000," *Journal of the American Medical Association* 292 (2004): 2372–8. See figure 3, 2376.

[20] Ibid., "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): 436–45; K. Ito, S. F. De Leon, and M. Lippmann, "Associations between Ozone and Daily Mortality: Analysis and Meta-Analysis," *Epidemiology* 16 (2005): 446–57; J. I. Levy, S. M. Chemerynski, J. A. Sarnat, "Ozone Exposure and Mortality: An Empiric Bayes Metaregression Analysis," *Epidemiology* 16 (2005): 458–68. The updated ozone staff report has not been posted on CARB's website as of this writing, but was emailed to CARB's "ozone standard stakeholders" list on July 18, 2005 and the updated version is dated July 18, 2005.

in the previous paragraph, this is equivalent to about a 0.88–1.04 percent increase in mortality per 0.019 ppm increase in daily eight-hour ozone, which is right in line with CARB's central estimate.

What CARB fails to mention is that all three of these studies, being meta-analyses, inherently suffer from publication bias, and that two provide evidence of that bias. In a 2005 study, M. L. Bell and colleagues compare their results to NMMAPS and show that the ozone effect estimated by meta-analysis is more than a factor of three higher than for NMMAPS. They attribute the discrepancy to the effect of publication bias on the meta-analytic results. K. Ito and colleagues likewise provide evidence of publication bias in their recent meta-analysis. An accompanying commentary on the three meta-analyses concludes:

> In the absence of NMMAPS or other multisite analyses, some observers might have taken the agreement of the meta-analyses as confirmation that the meta-analytic method was reliable. However, if our observational methods are all subject to the same biases, as meta-analyses are when they are derived from the same pool of studies, the agreement criterion is testing a narrow range of assumptions.[21]

In fact, CARB makes just this error by basing its mortality benefits on meta-analyses, rather than on studies that do not suffer from publication bias. In its updated staff report, CARB says of the new meta-analyses that "this estimate [of ozone mortality effects] is higher than the NMMAPS results; however, it is consistent with earlier meta-analyses of ozone time-series . . ."[22] Where CARB takes the new meta-analyses as providing confirmation of the agency's ozone-mortality estimates, they in fact show that CARB's mortality estimates are greatly inflated.

The studies discussed above address the effects of short-term daily fluctuations in ozone. A few studies have also looked at long-term ozone exposure and mortality. CARB's staff report cites two such studies on p. 10-55 and rightly notes that the evidence from them for long-term effects of ozone on mortality is weak and inconsistent.[23]

A study not cited by CARB provides additional evidence that long-term ozone exposure is not causing premature death, at least not at current levels. The Washington University–EPRI Veterans study assessed the relationship between air pollution and

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[21] S. N. Goodman, "The Methodologic Ozone Effect," *Epidemiology* 16 (2005): 430–35.

[22] California Air Resources Board, *Review of the California Ambient Air Quality Standard for Ozone*, July 18, 2005 update, p. B-12.

[23] The studies are D. E. Abbey, N. Nishino, W. F. McDonnell, et al., "Long-Term Inhalable Particles and Other Air Pollutants Related to Mortality in Nonsmokers," *American Journal of Respiratory and Critical Care Medicine* 159 (1999): 373–82, and 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): 1132–41.

mortality in fifty thousand male U.S. veterans.[24] The study population included men with preexisting high blood pressure, which could have made them more susceptible to ozone than the general population. The men were followed for twenty-one years after entry to the study, which concluded that ozone could be causing increased mortality but did not appear to have an effect until one-hour levels reached at least 0.15 ppm on at least eighteen days per year.[25] Such high levels are well above even the current federal one-hour standard, and suggest that there would be no long-term mortality benefits from reducing ozone below the level of that standard. The results from the long-term studies should also reduce one's confidence in the results of the short-term studies. If daily fluctuations in ozone were killing people, then one would expect to see an ozone-mortality association in the long-term studies.

CARB has clearly overstated the potential mortality benefits of ozone reductions. The evidence suggests that, at the very least, CARB's mortality-benefit claim should be revised downward by at least 50 percent. Taking account of model uncertainty, and of the anticorrelation between ozone and mortality in many cities, there is a good chance that reducing ozone will not prevent any premature deaths. About 90 percent of the health benefits of CARB's standard are due to reductions in premature mortality (see discussion in next section). If ozone is not causing premature death, then CARB's benefit estimate is too high by a factor of ten.

Health Effects of Long-Term Ozone Exposure

Aside from premature death, the risk of permanent health damage is among the most serious of harms supposed to be caused by ozone. Studies performed in Southern California based on ozone levels during the 1970s and '80s suggested that people who grow up in areas with high air pollution have reduced lung function when compared with those who grow up in areas with cleaner air. These studies were based on ozone levels in the worst areas of southern California, which at the time exceeded the federal one-hour and eight-hour ozone standards more than one hundred fifty days per year—much higher than current exceedance rates of, respectively, about twenty-five and one hundred days per year in the very worst areas. Thus, these past studies are not applicable to today's pollution levels.

A more recent California study provides evidence more relevant to pollution levels experienced today. CARB's Children's Health Study (CHS) has assessed a range of air-pollution health effects in thousands of children living in California.[26] The study began in 1992, and children in the study have been periodically assessed ever since. The

[24] 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): 41–73.

[25] The Veterans Study used the ninety-fifth percentile daily one-hour ozone level as its ozone exposure metric. The ninety-fifth percentile corresponds to roughly the eighteenth highest day of the year.

[26] CARB's description of the study can be found at http://www.arb.ca.gov/research/chs/chs.htm.

children live in twelve different California communities with a wide range of pollution levels, from very low up to the highest experienced anywhere in the nation. CHS results have been reported in a number of research publications, some of which I review below.

Long-Term Ozone Exposure and Lung Function. One group of children in the CHS was recruited in 1993 when they were in fourth grade and were followed through age eighteen.[27] The study reported no association between ozone levels and any measure of lung function.[28] Because these children were about ten years old when they entered the study, those who were born in the study areas had experienced California ozone levels from 1984 onward. Children living in the highest ozone areas—Lake Arrowhead and Riverside—had therefore grown up with ozone at levels exceeding the federal one-hour standard about one hundred fifty days per year for the first several years of their lives, and more than one hundred days per year up until their mid-teens. If such high ozone levels had no detectable effect on lung-function growth, then there is little chance that long-term exposure to ozone at levels below the federal one-hour standard could be affecting long-term lung function, and even less chance of an effect at levels below the current federal eight-hour standard.

Long-Term Ozone Exposure and Risk of Developing Asthma. CARB's staff report cites on p. 10-57 two California cohort studies of the risk of developing asthma due to ozone, McDonnell et al. (1999) and McConnell et al. (2002).[29] The McConnell study was part of the CHS and assessed asthma incidence relative to ozone levels during 1994–97 in twelve California communities with low to high ozone levels. The study reported that children playing three or more team sports (8 percent of all children) in the four communities with the highest ozone levels were 3.3 times as likely to develop asthma as similarly active children in four medium-ozone and four low-ozone communities.

However, overall, children were 30 percent *less* likely to develop asthma in the high-ozone communities based on one-hour ozone levels, and 20 percent less likely based on eight-hour ozone levels. The one-hour result was statistically significant, while the eight-hour result was just a hair short of statistical significance.[30] CARB reports these results incorrectly in its staff report on p. 10-58, claiming that asthma was "not higher" in

[27] W. J. Gauderman, E. Avol, F. Gilliland, et al., "The Effect of Air Pollution on Lung Development from 10 to 18 Years of Age," *New England Journal of Medicine* 351 (2004): 1057–67. In 1994, 1,759 fourth-graders entered the study. Due to attrition, 759 remained in the study through 2001.

[28] This was true whether ozone was measured based on peak daily one-hour or eight-hour averages. The lung-function measures were forced vital capacity (FVC), forced expiratory volume in the first second (FEV_1), and maximal mid-expiratory flow rate (MMEF).

[29] R. McConnell, K. T. Berhane, F. Gilliland et al., "Asthma in Exercising Children Exposed to Ozone: A Cohort Study," *Lancet* 359 (2002): 386-91; W. F. McDonnell, D. E. Abbey, N. Nishino et al., "Long-Term Ambient Ozone Concentration and the Incidence of Asthma in Nonsmoking Adults: The Ahsmog Study," *Environmental Research* 80 (1999): 110-21.

[30] The 95 percent confidence interval of the relative risk of developing ozone was 0.6–1.0. By standard criteria, the result would be considered statistically significant at the 5 percent level if the top of the range were below 1.0. Thus, the result is significant at just barely above the 5 percent level.

the high-ozone communities. In fact, it was lower.[31] Thus, taken at face value, the CHS asthma results suggest that higher ozone is overall associated with a *lower* risk of developing asthma.[32]

Even for the most active children, the CHS results are irrelevant to a discussion of CARB's eight-hour ozone standard, because the ozone level associated with asthma incidence is far above not only CARB's eight-hour ozone standard, but also either of the current federal ozone standards. For example, the four high-ozone communities in the CHS averaged 45 to 65 one-hour and 65 to 100 eight-hour exceedance days per year during 1994–97, when the CHS asthma study was performed. The medium- and low-ozone areas, in contrast, averaged between about zero and 40 one-hour exceedances and zero and 60 eight-hour exceedances. Thus, even taking the CHS asthma results at face value, even ozone levels that frequently exceed the federal one-hour standard do not increase the risk that children will develop asthma.

McDonnell et al. (1999) reported on air pollution and asthma incidence in Southern California as part of a long-term study of 3,091 nonsmoking Seventh Day Adventists. In this case, the study was based on ozone levels from 1973–92. The study reported that for men, a 0.010 ppm increase in average daily eight-hour ozone (measured from 9 a.m. to 5 p.m.) was associated with a 40 percent increase in the risk of developing asthma. Ozone was associated with a *lower* asthma risk for women, but the association was not statistically significant. This study was based on ozone levels far higher than those experienced in California today, and the study's results are therefore unlikely to be relevant to even the highest current ozone levels. In addition, as noted by CARB's staff, the fact that there was no association of ozone and asthma for women, despite a higher overall asthma incidence rate for women than men, "casts some doubt on these results."[33]

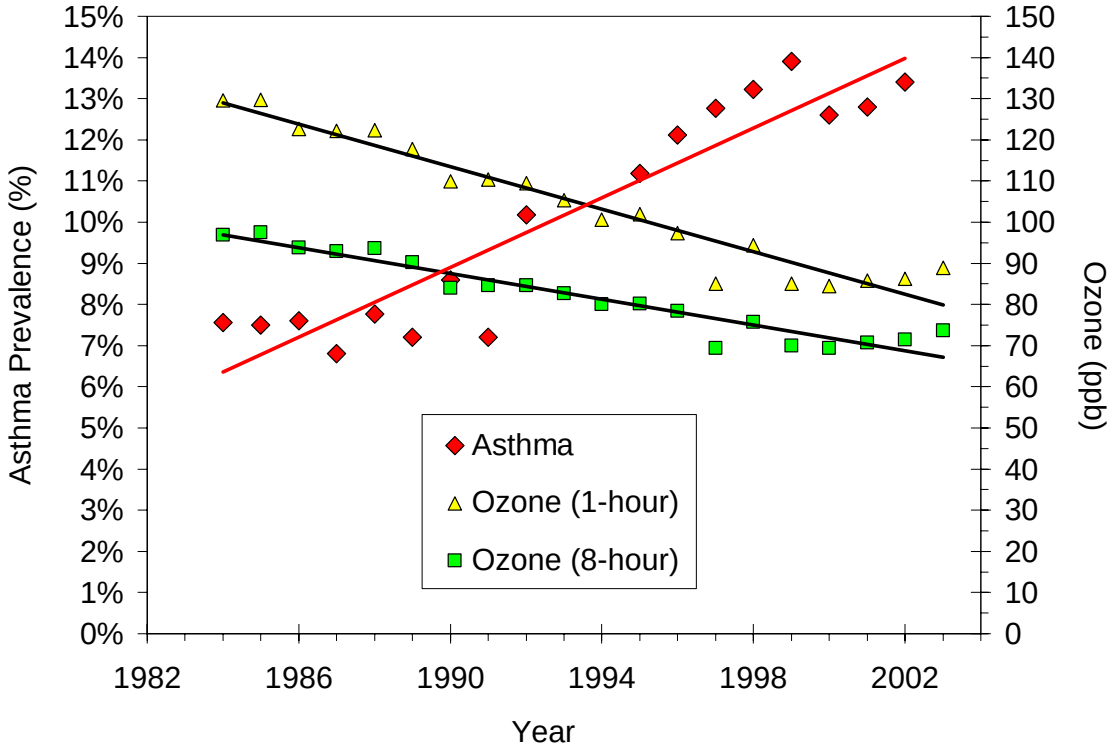
Ozone is not a plausible cause of asthma. Ozone levels have declined throughout California during the last twenty years, even as asthma prevalence has risen. For example, figure 5 compares the two trends since 1984. The ozone trend is based on the highest thirty daily ozone readings each year at each of sixty monitoring locations around the state that had continuous data over the entire period. The inverse relationship between trends in ozone and in asthma prevalence provides additional evidence that ozone is, at worst, not a significant factor in the development of asthma.

[31] In CARB's defense, McConnell et al. (2002) themselves describe asthma incidence as being "not higher" in the high-ozone communities in their *Lancet* paper. This is a misstatement of their own results, as the actual data they present in the paper show asthma incidence to be lower in the high-ozone communities.

[32] If the risk of developing asthma were 3.3 times greater for very active children (8 percent of all children) and 30 percent lower overall in the high-ozone areas, then for the other 92 percent of children the risk of developing asthma must have been 53 percent lower in the high-ozone areas.

[33] California Air Resources Board, *Review of the California Ambient Air Quality Standard for Ozone*, p. 10-57.

Figure 5. Trend in Asthma Prevalence vs. Ozone Levels in California



Notes: Ozone levels are an average of the thirty highest daily eight-hour and one-hour ozone values each year at each of sixty sites with continuous data for 1984–2003. Asthma prevalence is expressed as the percentage of the population that has asthma. The lines through each set of points are linear regression lines. Averages can sometimes hide varying trends by location. In this case, although slopes varied by location, ozone declined at all but one location during the last twenty years.

Sources: Ozone data are from California Air Resources Board, *California Ambient Air Quality Data 1980-2003*,. Asthma prevalence data were provided by staff at the California Department of Health Services.

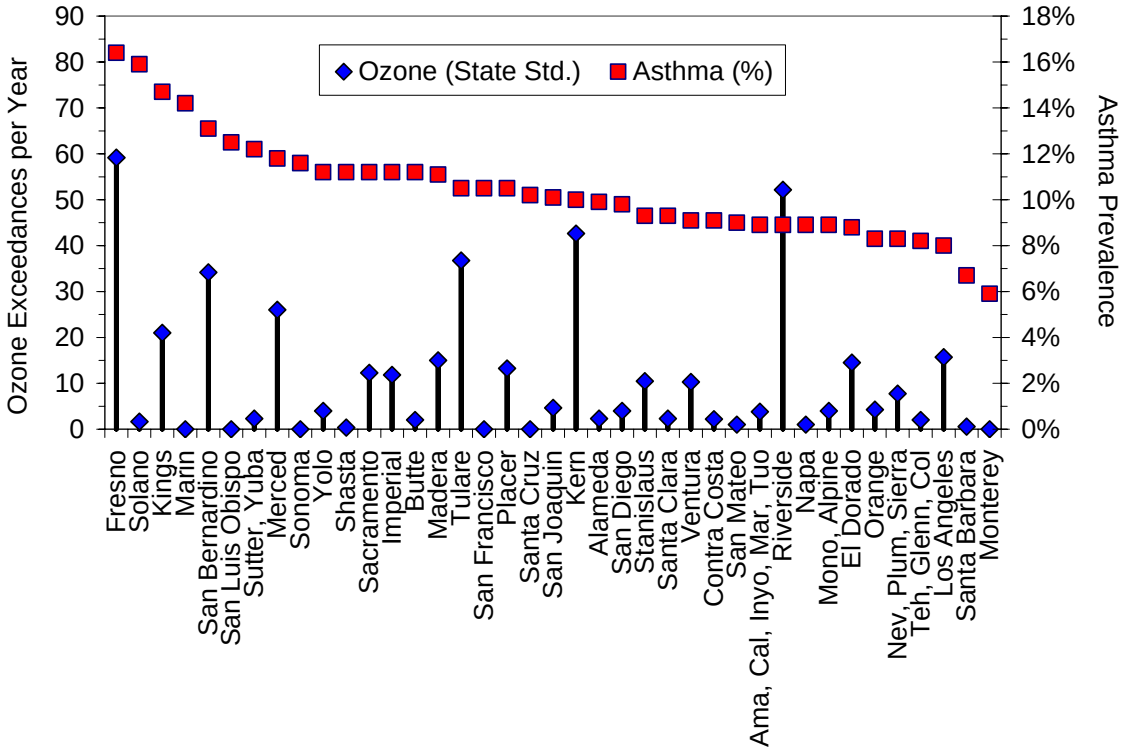
News and activist reports on air pollution often focus on Fresno County, because it has both high asthma prevalence and high air pollution, and they assert that the two are causally related.[34] We've just seen that a causal relationship between higher ozone and higher asthma prevalence is not plausible. Examination of county-by-county ozone levels and asthma prevalence likewise tells a different story than focusing solely on a single county with coincidentally high asthma and high ozone. Figure 6 shows there is no relationship between the number of ozone exceedance days per year and asthma prevalence by county. For example, Solano and Marin counties have high asthma but low

[34] See, for example, B. Anderson, "Asthma Steals Joys of Childhood," *Fresno Bee*, December 15, 2002; Fresno Bee, "Asthma in the Valley; More Research Is Needed into a Disease That Runs Rampant Here," *Fresno Bee*, October 4, 2004; Sacramento Bee, "Smog and Asthma: The Link—and Threat—Are Real," *Sacramento Bee*, May 6, 2003.

ozone. Kern and Riverside counties have high ozone and low asthma. Fresno happens to be high on both measures, but the weight of the evidence suggests this is a coincidence rather than a causal relationship.

While CARB's staff report doesn't explicitly claim that ozone is a cause of asthma, it is nevertheless vague about the evidence, and fails to present the strong evidence against ozone being a cause of asthma. The staff report also fails to note that the very low ozone levels addressed by CARB's standard are far below levels that could conceivably be associated with asthma incidence by even the most generous interpretation of the research literature.

Figure 6. Ozone Levels and Children's Asthma Prevalence by County, 2001



Notes: Number of days exceeding the California one-hour ozone standard (0.095 ppm) is an unweighted average for all monitoring sites in a county with data for 2001. Asthma prevalence by county for 2001 is the percentage of children ages five to fourteen who reported both having been previously diagnosed with asthma and experiencing asthma symptoms in 2001.

Sources: Ozone data for 2001 were retrieved from California Air Resources Board, *California Ambient Air Quality Data 1980-2003,*. Asthma data are from UCLA Center for Health Policy Research, *Asthma Symptom Prevalence in California in 2001* (Los Angeles: 2002), http://www.healthpolicy.ucla.edu/pubs/files/Asthma-by-county-052002.pdf.

Health Effects of Short-Term Ozone Exposure

Hundreds of epidemiological studies have assessed the extent to which ozone causes short-term health effects, from serious events, such as emergency room visits and hospitalizations, to milder problems, such as coughing and other respiratory symptoms, as well as absences from school due to illness.

Just as for estimates of ozone and mortality, CARB has overestimated other health effects by failing to account for model uncertainty and publication bias, by ignoring weaknesses in the studies selected for the health-effects estimates, and by omitting discussion of research that fails to find a relationship between ozone levels and health outcomes.

Hospital Admissions and Emergency Room Visits. To estimate reductions in hospital admissions due to ozone reductions, CARB uses a 1999 meta-analysis by Thurston and Ito.[35] But this study included only areas with cold climates.[36] Two studies of Medicare populations in the warmer climate of Birmingham, Alabama, did not find a statistically significant relationship between ozone and hospital admissions.[37] CARB did not use any California-based studies of hospital admissions for its benefit estimates, though the staff report does cite several studies in chapter 10, and notes that some of these studies did not find a relationship between ozone and hospital admissions.

As noted earlier, CARB used the WHO meta-analysis as part of its estimate of the mortality benefits of ozone reductions. This WHO study also included estimates of hospitalization rates due to ozone, but the size of the effect was much lower than the value from Thurston and Ito used by CARB and was also statistically insignificant.

Researchers from Kaiser Permanente studied the relationship between air pollution and emergency room visits and hospitalizations in California's Central Valley and reported that higher ozone was associated with a large, statistically significant *decrease* in serious health effects.[38] CARB sponsored this study and put out a press release when it was released, yet does not discuss or cite the study in its staff report.[39]

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[35] California Air Resources Board, *Review of the California Ambient Air Quality Standard for Ozone*, p. B-11; G. D. Thurston and K. Ito, "Epidemiological Studies of Acute Ozone Exposures and Mortality," *Journal of Exposure Analysis and Environmental Epidemiology* 11 (2001): 286-94.

[36] J. I. Levy, T. J. Carrothers, J. T. Tuomisto, et al., "Assessing the Public Health Benefits of Reduced Ozone Concentrations," *Environmental Health Perspectives* 12 (2001): 9–20; Thurston and Ito, "Epidemiological Studies of Acute Ozone Exposures and Mortality."

[37] Cited in ibid.

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

[39] California Air Resources Board, "Hospitalizations and Emergency Room Visits Increase Following High Particulate Matter Episodes, Study Finds," press release, February 24, 2003,

In addition to the epidemiological results, inspection of data from around the country, including California, shows that emergency room visits for asthma are lowest in July and August, when ozone is highest and children spend more time outside than at other times of the year.[40]

Respiratory Symptoms. A number of studies have assessed respiratory symptoms in children residing in California's high- and low-ozone areas, and several have reported a lack of association between ozone levels and respiratory symptoms.[41] CARB's Children's Health Study did not find a relationship between long-term ozone levels and self-reported respiratory symptoms.[42] A study of asthmatic children in Los Angeles reported a statistically significant *decrease* in coughing associated with higher ozone levels.[43] A 1994 study of asthmatic, wheezy, and healthy children in high-ozone areas of California

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http://www.arb.ca.gov/newsrel/nr022403.htm. CARB's press release mentions results for particulate matter, which was positively associated with health effects, but omits the ozone results.

[40] For data on asthma symptoms and hospital admissions by month, see, for example, 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; Spokane Regional Health District, *Asthma in Spokane County* (Spokane, Washington: 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; 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* (Michigan Department of Community Health, undated), http://www.michigan.gov/documents/Childhood_Asthma_6549_7.pdf.

[41] See, for example, R. J. Delfino, R. S. Zeiger, J. M. Seltzer, et al., "The Effect of Outdoor Fungal Spore Concentrations on Daily Asthma Severity," *Environmental Health Perspectives* 105 (1997): 622–35; W. S. Linn, D. A. Shamoo, K. R. Anderson, et al., "Short-Term Air Pollution Exposures and Responses in Los Angeles Area Schoolchildren," *Journal of Exposure Analysis and Environmental Epidemiology* 6 (1996): 449–72; B. Ostro, M. Lipsett, J. Mann, et al., "Air Pollution and Exacerbation of Asthma in African-American Children in Los Angeles," *Epidemiology* 12 (2001): 200–208. Despite the lack of an association between ozone and asthma symptoms in Ostro et al., ozone was associated with an increase in asthma medication use.

[42] R. McConnell, K. Berhane, F. Gilliland, et al., "Prospective Study of Air Pollution and Bronchitic Symptoms in Children with Asthma," *American Journal of Respiratory and Critical Care Medicine* 168 (2003): 790–97; J. M. Peters, E. Avol, W. Navidi, et al., "A Study of Twelve Southern California Communities with Differing Levels and Types of Air Pollution. I. Prevalence of Respiratory Morbidity," *American Journal of Respiratory and Critical Care Medicine* 159 (1999): 760–67. In the McConnell et al. study, ozone was associated with increased bronchitis symptoms in asthmatic children in a single-pollutant model, but the ozone effect became statistically insignificant after adjustment for confounding by other pollutants.

[43] Ostro, Lipsett, Mann, et al., "Air Pollution and Exacerbation of Asthma in African-American Children in Los Angeles."

reported inconsistent associations between ozone and respiratory symptoms.[44] Asthmatic children had increased symptoms on higher-ozone days, while wheezy children had increased symptoms on lower-ozone days.

The staff report makes special mention on p. 10-7 of a study of asthma symptoms in 271 children in southern New England, calling it "one of the largest and best conducted studies" and asserting that it "provides the strongest evidence for effects of ozone independent of $PM_{2.5}$" on asthma symptoms.[45] The study reported that for asthma sufferers who used medication to control their asthma, a 0.05 ppm increase in one-hour ozone levels was associated with a 35 percent increase in the risk of wheezing and a 47 percent increase in the risk of chest tightness. Symptoms of persistent cough and shortness of breath were also associated with higher ozone. Ozone was not associated with any symptoms in asthmatics who were not on medication.

Despite CARB's laudatory review, technical aspects of the study's statistical analysis reduce the reliability of its results. For example, instead of using the nearest pollution monitor to represent ozone exposure for a given child, the researchers averaged pollution levels at all monitors in the study area, which were spread over a few thousand square miles of Connecticut and western Massachusetts. This creates the likelihood of large errors in ozone exposures assigned to the children in the study.

Furthermore, the statistical model used for the study inadequately controlled for weather. For example, the model included only same-day weather, but not weather during the previous few days, and temperature was the only weather variable in the model. The study also did not control for day of the week and season, which could confound the results. For example, asthma symptoms rise in September, independent of pollution levels.[46]

The study also reported greater effects based on one-hour ozone levels than for eight-hour levels. For example, while one-hour ozone was associated with increased wheezing, eight-hour ozone was not. For other symptoms, eight-hour ozone was associated with smaller risks than one-hour ozone. CARB's staff report claims misleadingly on p. 10-7 that "both daily maximum 1-hr and 8-hr concentrations were similarly related to symptoms, including chest tightness and shortness of breath."

Like estimates of ozone and mortality, epidemiological estimates of other short-term ozone health effects likely suffer from publication and model-selection biases, but CARB's staff report does not mention or attempt to account for these effects when

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[44] E. L. Avol, W. C. Navidi, E. B. Rappaport, et al., "Acute Effects of Ambient Ozone on Asthmatic, Wheezy, and Healthy Children," *Research Report /Health Effects Institute* (1998): 1–18; discussion 19–30.

[45] Gent, Triche, Holford, et al., "Association of Low-Level Ozone and Fine Particles with Respiratory Symptoms in Children with Asthma."

[46] N. W. Johnston, S. L. Johnston, J. M. Duncan, et al., "The September Epidemic of Asthma Exacerbations in Children: A Search for Etiology," *Journal of Allergy and Clinical Immunology* 115 (2005): 132–38.

estimating the ostensible benefits of additional ozone reductions. From among all the estimates in the literature, CARB also selected studies reporting relatively large ozone effects when performing its benefit estimates for ozone reductions. As a result, CARB's estimates likely overstate the health benefits of ozone reductions.

School Absences. Based on CARB's estimates, going from current ozone levels to attainment of its eight-hour standard would reduce school absences by nearly 9 percent—a reduction of 3.7 million total school absences per year, or 0.54 absence days per student.[47] The incremental benefit of CARB's standard alone would be nearly half this amount, or a reduction of 0.24 absence days per student.

Whatever the benefits of ozone reductions in terms of school absences, they would presumably apply to reductions in absences related to respiratory illnesses, rather than to, say, gastrointestinal illnesses or non-illness-related absences. Data collected in the Children's Health Study suggest that respiratory illness is a factor in about 35 percent of school absences.[48] If so, then CARB implicitly predicts that attaining its eight-hour standard will reduce respiratory-related absences by 26 percent.[49]

CARB's estimate of the decrease in school absences due to ozone reductions is not credible. First, even without examining the merits of the estimate itself, comparison with CARB's other health-effects estimates raises concerns about consistency. CARB predicts that reducing ozone is seventeen and twenty-two times as effective in reducing respiratory-related school absences as in reducing, respectively, "restricted-activity days" and respiratory hospital admissions. The degree of sickness necessary to cause an absence from school would presumably fall somewhere within the range of the other two illnesses, making CARB's estimate for ozone and school absences inconsistent with the estimates for other illnesses.

Second, CARB ignores the biological implausibility of the results in Gilliland et al.—the 2001 study on which CARB based its estimates of school absence.[50] Gilliland et al. reported that a 0.020 ppm increase in eight-hour ozone levels was associated with an 83 percent increase in school absences due to respiratory causes. However, the apparent effects of ozone were due mainly to levels from one or two weeks ago, rather than during

[47] According to CARB's staff report, going from current ozone levels to statewide attainment of its new eight-hour standard would eliminate 3.7 million absence days per year. CARB also estimates that students are absent an average of six schooldays per year. There are about 6.9 million students in California's primary and secondary schools (including public and private schools), and the average school year is about 180 days. Based on these values, CARB implicitly estimates that attaining its standard will reduce school absences by nearly 9 percent.

[48] F. D. Gilliland, K. Berhane, E. B. Rappaport, et al., "The Effects of Ambient Air Pollution on School Absenteeism Due to Respiratory Illness," *Epidemiology* 12 (2001): 43–54.

[49] If, on the other hand, ozone reductions are equally effective in reducing the risk of absence due to any illness, then attaining CARB's standard would reduce illness-related absences by 16 percent.

[50] Gilliland, et al., "The Effects of Ambient Air Pollution on School Absenteeism Due to Respiratory Illness."

the last few days. Time spent outdoors, which would have increased ozone exposures, was associated with fewer school absences. When the study assessed particulate matter under ten microns in diameter (PM_{10}) it concluded that PM_{10} was associated with a large increase in non-illness-related absences, but not with absences due to illness. An increase of ten micrograms per cubic meter in PM_{10} was associated with a larger effect on non-illness-related absences than was an 0.020 ppm increase in ozone on respiratory-related absences.

Taken together, these results are biologically implausible and suggest that the apparent effect of ozone on school absences was a statistical anomaly, possibly due to failure to control adequately for season, rather than a real cause and effect relationship.

Third, CARB's staff report cites only the Gilliland et al. (2001) study for its estimate of school absences related to air pollution levels. In fact, two other studies addressed this issue using the same California Children's Health Study data as Gilliland et al., but reported no statistically significant association between daily ozone levels and school absences.[51] One reported an association between long-term ozone levels and school-absence rates, while the other did not. In the former case, the ozone effect was only about one-fourth the size of short-term ozone effect reported by Gilliland et al.

CARB does not mention either of these studies in its staff report, even though both used Children's Health Study data and were authored by researchers who participated in other CHS analyses, including the Gilliland et al. study. And even the more recent of the two studies was published several months before CARB released its final staff report.

These two studies, Berhane and Thomas (2002) and Rondeau et al. (2005), provide additional information that casts doubt on the claim of an air pollution–school absence association. For example, both show that the apparent effect of short-term ozone levels on respiratory absences increases as the "lag-time"—that is, the number of days of ozone exposure before a school-absence day—included in the statistical model increases. When looking at only the last five days of ozone exposures before an absence, Rondeau et al. reported that ozone was actually associated with a small, statistically insignificant *decrease* in school absences. It was associated with an insignificant increase in absences once the ozone-exposure lag-time was increased to fifteen days, and the ozone effect increased a bit more at a thirty-day lag, though the effect was still small and far from statistical significance.

Berhane and Thomas reported no ozone association with illness-related absences over a fifteen-day lag-period, but did report a nearly significant ($p = 0.075$) association for a thirty-day lag-period. As noted earlier, it seems implausible that ozone exposures

[51] K. Berhane and D. C. Thomas, "A Two-State 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.

from weeks ago would have a greater effect on school absences than exposures during the last few days.

All three of the studies discussed here were based on data collected in twelve California communities. While two present the average absence rate versus ozone-exposure lag-time, Berhane and Thomas also presents the results for each of the twelve communities individually, revealing great variation among them. For example, ozone exposures from up to a few days ago are associated with a large increase in absences in a few communities, a large decrease in others, and little change in still others. Ozone exposures from one or two or three weeks ago had a similar range of apparent effects. These wild variations among communities in health effects versus time-since-ozone-exposure don't appear to be biologically plausible, and once again suggest that ozone is not actually causing school absences.

Overall, the evidence suggests that if ozone is causing any school absences, the effect is, at worst, much smaller than CARB claims. The inconsistency and biological implausibility of many of the epidemiological results suggest that school absences may be unrelated to ozone levels.

Laboratory Studies

The discussions above address the results of epidemiological studies. It is also possible to assess ozone's effects under controlled conditions in laboratory studies with human volunteers. The volunteers are exposed to varying levels of air pollution or clean air, perhaps while exercising, and are given lung-function tests to assess whether pollution causes any change in respiratory performance.

Laboratory studies have the advantage of being able to establish a cause and effect relationship between pollution exposure and the results of lung-function and other physiological tests. But laboratory studies also have limitations. Because it would be unethical put people at risk of serious harm, laboratory studies can only assess relatively mild effects, for example, changes in the maximum amount of air a person can blow out in one second, or subjective symptoms, such as pain while breathing. Of course, laboratory studies can't be used to assess the extent to which air pollution kills or sends people to the hospital. The studies generally also include small samples—usually no more than a few dozen people—and there is uncertainty in the applicability of the particular laboratory conditions to real-world air-pollution exposures in terms of pollution levels, mixture of pollutants present, level of physical activity, and length of exposure.

In general, for exposures of one to two hours with exercise, these studies report small average reductions in lung function at ozone levels at around 0.12 ppm or above. Ozone levels as low as 0.08 ppm are associated with reductions when people are exposed

for more than five hours while exercising nearly continuously.[52] Although the average reduction in lung function was small in these studies—scores on various lung-function tests declined an average of a few to several percent—a few people experienced larger reductions in lung test scores. Effects of these short-term exposures were temporary, and lung function returned to normal within a day.

A full review of the dozens of controlled exposure studies is beyond the scope of this paper; however, the results of specific studies have been reviewed by the EPA and CARB and by various commenters on the EPA's and CARB's reviews of the literature.[53] Details of methodologies and specific results aside, a key question with these studies is the extent to which they are relevant to the setting of air-pollution standards, particularly at the low ozone levels addressed by the current federal eight-hour standard or CARB's standard. There are two key issues:

- Ozone levels measured at fixed monitoring are much higher than the personal ozone exposures people actually experience.

- Laboratory studies use ozone-free air as the reference exposure, rather than a background ozone level.

Personal vs. Ambient Ozone Exposure. Comparisons of ozone exposures measured by personal monitors, with ambient ozone measured at the fixed ozone monitoring sites used for regulatory compliance, show that personal exposures are much lower than ambient

[52] D. H. Horstman, L. J. Folinsbee, P. J. Ives, et al., "Ozone Concentration and Pulmonary Response Relationships for 6.6-Hour Exposures with Five Hours of Moderate Exercise to 0.08, 0.10, and 0.12 ppm," *American Review of Respiratory Disease* 142 (1990): 1158–63; S. M. Horvath, J. F Bedi, D. M. Drechsler, and R. E. Williams, "Alterations in pulmonary function parameters during exposure to 80 ppb ozone for 6.6 hours in healthy middle aged individuals (1991), in R. L. Berglund, D. R. Lawson, and D. J. McKee, eds. Tropospheric ozone and the environment: papers from an international conference, March 1990, Los Angeles. Air & Waste Management Association, pp. 59-70; W. F. McDonnell, H. R. Kehrl, S. Abdul-Salaam, et al., "Respiratory Response of Humans Exposed to Low Levels of Ozone for 6.6 Hours," *Archives of Environmental Health* 46 (1991): 145–50.

[53] In addition to CARB's staff report, also see Environmental Protection Agency, *Air Quality Criteria for Ozone and Other Photochemical Oxidants* (Washington, D.C., July 1996); S. R. Hayes, *Initial Comments on California's Draft Ozone Staff Report* (Emeryville, California: Environ International; prepared for the American Petroleum Institute, August 31, 2004), http://www.arb.ca.gov/research/aaqs/ozone-rs/comments/api-wspa-9.pdf; J. Heuss and D. Kahlbaum, *Comments on June 21, 2004 Public Review Draft 'Review of the California Ambient Air Quality Standard for Ozone* (Air Improvement Resource; prepared for the Alliance of Automobile Manufacturers, September 1, 2004), http://www.arb.ca.gov/research/aaqs/ozone-rs/comments/alliance9.pdf; A. Lefohn, *Comments on the California Ambient Air Quality Standard for Ozone Document (CAAQSOD)* (Helena, Montana: ASL Associates; prepared for the American Petroleum Institute, August 31, 2004), http://www.arb.ca.gov/research/aaqs/ozone-rs/comments/api-wspa-9.pdf; S. H. Moolgavkar, *Comments on the 'Review of the California Ambient Airborne Standard for Ozone'* (Alexandria, Virginia: Sciences International; prepared for the Engine Manufacturers Association, September 1, 2004), http://www.arb.ca.gov/research/aaqs/ozone-rs/comments/ema-9.pdf; S. H. Moolgavkar, *Review of Chapter 10* (Bellevue, Washington: September 24, 2004), http://www.arb.ca.gov/research/aaqs/ozone-rs/comments/ema-9.pdf.

levels, even when comparing only outdoor personal to outdoor ambient exposures. Thus, for example, when ambient ozone is 0.08 ppm, personal exposures outdoors are typically on the order of 40–60 percent lower than the ambient level.[54] This means that a laboratory study of the effects of 0.08 ppm ozone is not representative of 0.08 ppm in ambient air, but of ambient levels more like 0.12 to 0.18 ppm. When time spent indoors is taken into account, personal ozone exposures are even further below ambient levels. Evidence on personal versus ambient ozone exposures includes the following:

- A 1997 study by L. J. Liu et al. found that a group of forty children and adults in Alpine, California, experienced average personal ozone exposures 75 percent lower than ambient levels. The authors attributed the difference mainly to time spent indoors and the lower ozone levels typical of indoor environments. However, on any given day, even the highest personal ozone exposure was usually well below the ambient ozone level.[55] Since the study was performed during spring and fall in an area with little rain and a mild climate, presumably some of these people spent a great deal of time outdoors. Nevertheless, their personal ozone exposures were much lower than ambient. Out of about 2,100 person-days measured, there were only three person-days in which personal ozone exposure exceeded 0.07 ppm.[56] This is despite the fact that 27 out of the 100 days studied had ambient ozone exceeding 0.07 ppm. In probabilistic terms, for each day in which ambient ozone exceeded 0.07 ppm, an average of only 1 in 190 people (0.53 percent) was actually exposed to ozone exceeding 0.07 ppm.[57]

- A group of trained technicians in eastern Los Angeles County wore personal

[54] Several factors probably contribute to lower outdoor personal exposures when compared to ambient monitors. These include the fact the ambient monitors are often placed several feet above typical human head-height to avoid interferences from people and surfaces near the ground. However, ozone deposition on surfaces (such as clothing or the ground) can reduce ozone levels in air that people actually breathe. Ozone levels also tend to be lower near roads, due to ozone destruction by nitric oxide emitted by vehicles. Finally, there is evidence that the equipment used for regulatory ozone monitoring gives readings that are biased high. A. R. Leston, W. M. Ollison, C. W. Spicer, et al., "Potential Interference Bias in Ozone Standard Compliance Monitoring," Proceedings of the AWMA Specialty Conference, VIP-126-CD, Symposium on Air Quality Measurement Methods and Technology, Research Triangle Park, N.C., Air & Waste Management Association, Pittsburgh, 2004.

[55] L. J. Liu, R. Delfino and P. Koutrakis, "Ozone Exposure Assessment in a Southern California Community," *Environmental Health Perspectives* 105 (1997): 58–65. On nine out of the one hundred study days, the most exposed participant had a personal exposure that was actually somewhat higher than ambient levels, though these tended to be days with relatively low ambient levels.

[56] These ozone levels are based on twelve-hour rather than eight-hour averages. The study did not report eight-hour averages. For any given eight-hour-average ozone level, the twelve-hour average would be expected to be somewhat lower.

[57] It is, of course, possible that this group of fifty people is not representative of the typical resident of Alpine. On the other hand, the results of this study are consistent with the other studies discussed in this section.

ozone monitors and performed scripted activities, such as walking outdoors near or away from a roadway, sitting in a backyard, driving with windows open or closed, performing normal household activities indoors, and so forth, during specific times of the day.[58] The technicians amassed a total of twenty-one person-days of measurements. Hourly outdoor personal exposures averaged about 40 percent lower than hourly ambient ozone levels reported at the nearest monitors.

- A year-long study of 169 children in Upland and the Crestline area, both high-ozone regions in San Bernardino County, California, reported that personal ozone exposures during the ozone season (May–September) averaged 61 and 58 percent below the respective ambient levels in the two areas.[59] This ratio includes total personal exposure (outdoor, indoor, and in-vehicle). The study did not report the ratio for outdoor-personal to outdoor-ambient exposure, although the ratio of personal-to-ambient exposure was greater during the summer than the winter, reflecting the greater amount of time children spent outdoors during the summer.

- A dozen asthmatic children in Alpine, California experienced average personal ozone exposures 75 percent lower than ambient levels.[60] This study likewise did not report an outdoor-personal to outdoor-ambient ratio.

- In a study of thirty-six children in Tennessee, those in the top 25 percent of time-spent-outdoors nevertheless experienced personal ozone exposures 80 percent lower than ambient levels.[61]

- Outdoor workers in Mexico City experienced average personal ozone exposures 60 percent lower than ambient levels in a study of thirty-nine shoe-cleaners.[62] All ozone exposures in this study took place outdoors.

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[58] T. Johnson, K. Clark, K. Anderson, et al., "A Pilot Study of Los Angeles Personal Ozone Exposures during Scripted Activities," *Measurement of Toxic and Related Air Pollutants* (Research Triangle Park, NC: Air and Waste Management Association, May 7–9, 1996)..

[59] A. S. Geyh, J. Xue, H. Ozkaynak, et al., "The Harvard Southern California Chronic Ozone Exposure Study: Assessing Ozone Exposure of Grade-School-Age Children in Two Southern California Communities," *Environmental Health Perspectives* 108 (2000): 265–70.

[60] R. J. Delfino, B. D. Coate, R. S. Zeiger, et al., "Daily Asthma Severity in Relation to Personal Ozone Exposure and Outdoor Fungal Spores," *American Journal of Respiratory and Critical Care Medicine* 154 (1996): 633–41.

[61] K. Lee, W. J. Parkhurst, J. Xue, et al., "Outdoor/Indoor/Personal Ozone Exposures of Children in Nashville, Tennessee," *Journal of the Air and Waste Management Association* 54 (2004): 352–59.

[62] M. S. O'Neill, M. Ramirez-Aguilar, F. Meneses-Gonzalez, et al., "Ozone Exposure among Mexico City Outdoor Workers," *Journal of the Air and Waste Management Association* 53 (2003): 339–46.

Overall, these studies indicate that personal ozone exposures, even while outdoors, are much lower than ambient ozone levels.

A second concern with the laboratory studies is that they compare lung function when volunteers breathe, say, 0.08 ppm ozone to lung function when breathing ozone-free air. But this is not a real-world situation. Even without any human activity in California, there would still be some ozone in the air due to a combination of natural ozone-forming emissions, transport of human-caused and natural ozone- and ozone-forming pollution from other areas of the world, downward transport of ozone from the stratosphere, and cloud-related electrical activity.[63]

The background level of ozone is a matter of controversy. For the purposes of its analysis, CARB assumed 0.04 ppm as the daily peak level. If this is a realistic background, then comparing ozone-free air with 0.08 ppm ozone involves a change in exposure that is twice as large as the real-world change in going from background to 0.08 ppm. There is only one published study of changes in lung function at ozone levels of zero, 0.04, and 0.08 ppm.[64] This study, by W. C. Adams, exposed college students to these ozone levels with vigorous exercise for 6.6 hours. There was no statistically significant difference in average lung-function tests between the zero and 0.04 ppm ozone exposures. Average lung-function test scores declined about 5 percent between the 0.04 ppm to 0.08 ppm exposures, though this difference appeared only after six hours of exposure. This ozone effect is smaller than the effect of going from zero to 0.08 ppm ozone, and suggests that the failure to use a realistic background-ozone concentration to represent "clean air" can cause laboratory studies to overstate changes in lung function due to real-world ozone exposures.

CARB's main justification for its standard is that it will prevent discomfort and temporary lung-function reductions observed in laboratory studies at ozone levels of 0.08 ppm. The staff report states on p. 2-9 "our recommendation for the eight-hour standard is based primarily on the chamber studies that have been conducted over the last 15 years, supported by the important health outcomes reported in many of the epidemiologic studies." However, taking account of the relationships between ambient and personal ozone exposures and background ozone, the laboratory studies do not provide evidence that even the current federal eight-hour ozone standard is necessary. The lowest-exposure laboratory studies have generally used an exposure range from zero to 0.08 ppm. If personal outdoor exposures are typically 40 percent below ambient, then in terms of ambient ozone levels, the zero to 0.08 ppm lab studies correspond to a real-world personal exposure range of something like 0.025 to 0.13 ppm—a range and peak level of

[63] Natural sources of ozone and ozone-forming emissions are discussed in the next section.

[64] W. C. Adams, "Comparison of Chamber and Face-Mask 6.6 Hour Exposures to Ozone on Pulmonary Function and Symptom Responses," *Inhalation Toxicology* 14 (2002): 745–64.

exposure far too large to be relevant to either the current federal or new California eight-hour ozone standards.[65]

If CARB wishes to justify its current standard based on laboratory studies, it would need evidence of adverse effects when comparing people's lung function after breathing 0.025 and 0.045 ppm ozone for several hours with vigorous exercise. This is the range in personal exposure that would be expected in going from an ambient background of 0.04 ppm to an ambient level of 0.075 ppm. Adams did not find any statistically significant change in average lung function in his laboratory study comparing zero and 0.04 ppm ozone exposures, even though the participants were exposed for more than six hours while exercising nearly continuously and strenuously. This is the only study to assess such low ozone exposures in a laboratory setting. It suggests that people are unlikely to suffer ill effects due to personal exposures that would be associated with ambient levels in the vicinity of CARB's standard. Ambient ozone at the current federal standard would be roughly equivalent to a personal exposure of about 0.05 ppm. Once again, this is unlikely to result in adverse effects, and we can therefore conclude that even the current federal eight-hour ozone standard is more than protective against the health effects that appeared to be of most concern to CARB for determining the level of the eight-hour standard.

CARB's staff report devotes a few pages in chapter 7 to a discussion of personal ozone exposures, but fails to draw any conclusions about what the personal exposure results suggest about real-world ozone exposure patterns and the implications of these patterns for laboratory studies of ozone's effects. In summarizing the personal exposure data, the agency's only conclusion on the subject is that "outdoor ozone exposures are more reflective of peak exposures, which may be more relevant in determining health impacts."[66]

Outdoor exposures *are* more relevant to determining peak exposures, but what matters is the level of these peak exposures. CARB avoids coming to terms with the finding that personal ozone exposures, even when outdoors, are significantly lower than ambient levels measured at fixed monitoring sites. The staff report also fails to discuss or cite the Tennessee, Mexico, or Los Angeles County studies discussed above. As in other sections of the report, CARB thus creates the appearance of having evaluated the weight of the evidence, while in fact the agency has omitted key studies and has, through vagueness or omission, led readers to draw incorrect conclusions about the nature and weight of the evidence.

The results of personal-exposure studies create an interesting paradox. The personal-exposure studies suggest that controlled laboratory studies overstate the health effects of any given ambient ozone level, due to the fact that laboratory studies use levels

[65] I derived this range by reducing the presumed ambient background of 0.04 ppm by 40 percent, and multiplying 0.08 ppm by 1.66 (which is the inverse of reducing 0.13 ppm by 40 percent).

[66] California Air Resources Board, *Review of the California Ambient Air Quality Standard for Ozone*, p. 7-138.

keyed to ambient monitoring rather than personal exposure. However, they suggest just the opposite conclusion when compared with epidemiological studies. The epidemiological studies are based on ambient ozone measured at fixed monitoring sites. But if a given personal exposure is 40 percent lower than a given ambient level, the health effects reported in epidemiological studies are occurring at much *lower* personal ozone exposures than one would assume based on ambient levels. If the epidemiological studies are detecting real cause and effect relationships between ozone and health, this is a genuine paradox. On the other hand, the paradox would be resolved to the extent that the epidemiological results represent chance statistical correlations due to methodological problems, or overestimates due to publication bias.

CARB Estimates Most Health Benefits Come from Reducing Ozone below Levels that Already Comply with CARB's Standard

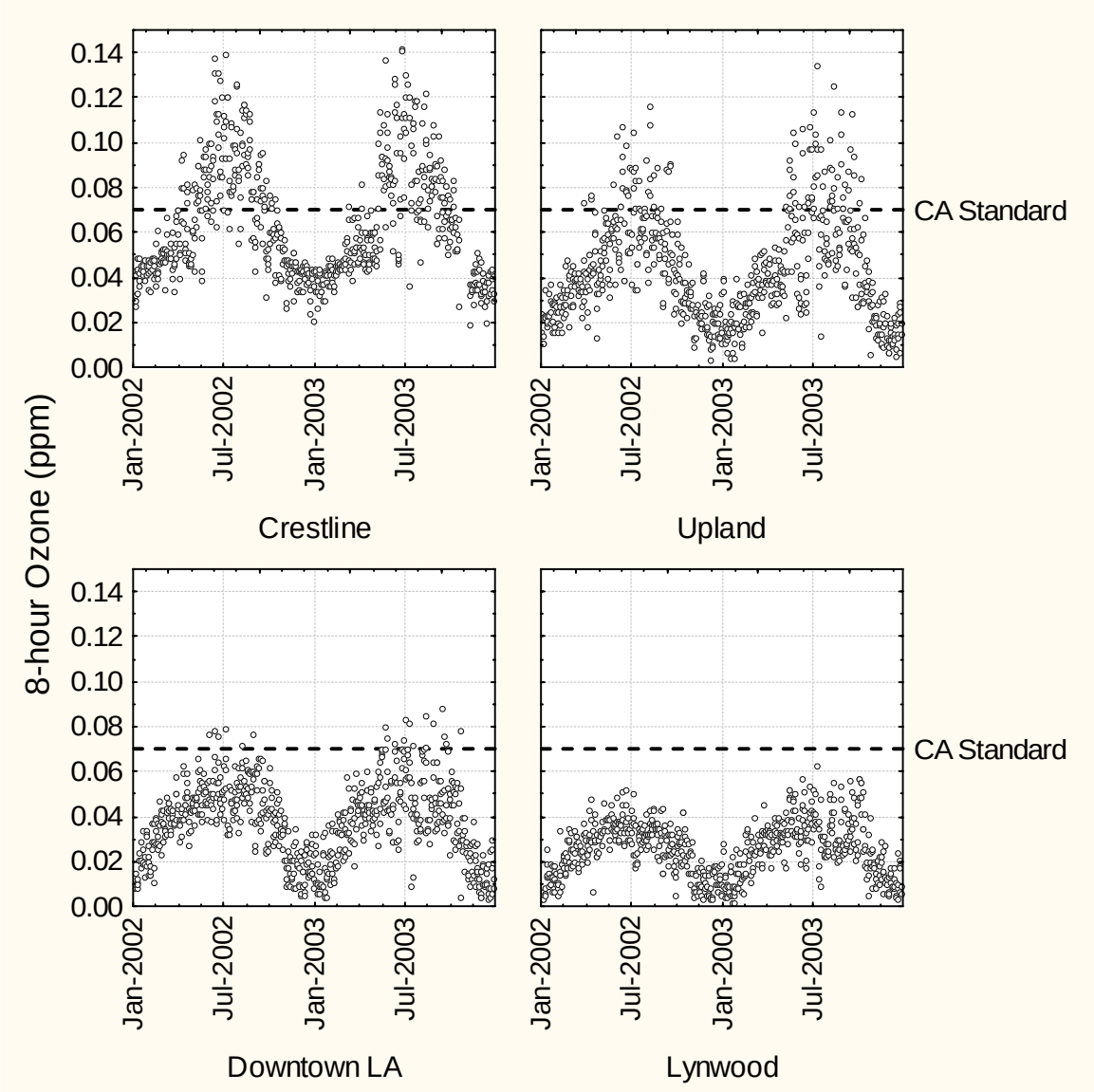
Because CARB has adopted an ozone standard set at 0.070 ppm, one might expect that the presumed benefits would be due to reducing ozone from levels above the standard down to the standard. But the vast majority of the benefits CARB predicts actually come from reducing ozone within levels that already comply with the standard. CARB estimates that 76–86 percent of the predicted benefits fall into this category.

Figure 7 shows why. The figure displays daily peak eight-hour ozone levels from January 2002 to December 2003 at four monitoring locations in South Coast (the Los Angeles metropolitan area). Crestline has the worst ozone in South Coast, while Lynwood has among the lowest levels in the area. The dashed horizontal line marks CARB's standard. As the graph shows, ozone is already below CARB's standard but above 0.04 ppm on most days, even at Crestline. For South Coast to attain the standard, ozone on the worst day at the worst site—Crestline—would have to be reduced to the standard. CARB sensibly reasons that measures that reduce ozone on the worst day at the worst site would also reduce ozone at other sites and other days, including days and locations for which ozone already falls below the standard.[67]

CARB claims to be setting its ozone standard at a level that will protect public health with an adequate margin of safety. If so, then CARB's incremental health-benefit estimate should be reduced by 80 percent, even before taking account of the likelihood that the health benefits of lower ozone are much smaller than CARB claims. Data from personal-exposure studies support this view as well. If there are no effects on lung function in going from an ambient level of 0.04 ppm to an ambient level of 0.075 ppm—a personal-exposure range of 0.025 to 0.045 ppm—as suggested by laboratory studies, then there certainly won't be any effects in going from 0.04 ppm ambient up to the lower ozone levels typical of most days in most places.

[67] CARB assumes a uniform background one-hour ozone level of 0.04 ppm. This background is a lower limit on how much ozone can be reduced for the purposes of CARB's health-benefit calculations. After calculating the ozone reduction necessary on the worst day and the worst site in order to attain the proposed standard, CARB reduces ozone proportionally for other sites, and for other days at the worst site in order to predict total health benefits due to attaining the standard. See page 10-23 of CARB's staff report for details.

Figure 7. Peak Daily Eight-Hour Ozone Levels at Four South Coast Monitoring Locations, 2002–03



Notes: For each monitoring location, a point on the graph represents the highest eight-hour-average ozone reading on a given date. The dashed horizontal line marks California's eight-hour ozone standard, so readings above the dashed line represent days in which ozone exceeded the standard. Lynwood is on the west side of Los Angeles near the coast, while Crestline is the easternmost location.

Source: California Air Resources Board, *California Ambient Air Quality Data 1980-2003*.

Attempting to Attain CARB's Ozone Standard Will Harm Californians

Even attaining the current federal eight-hour ozone standard will be difficult in some areas of California. Attaining CARB's standard will be harder still. CARB's eight-hour standard is set at 0.070 ppm, and no exceedances are allowed. But even if no one lived in California, it is likely that background ozone levels would sometimes exceed 0.070 ppm in some parts of the state. If so, then statewide attainment of CARB's standard is impossible.

The measures necessary to reduce ozone are costly. People ultimately pay for ozone-reduction requirements in the form of higher prices for the goods and services they purchase, in lower wages, and in reductions in their freedom to live their lives in ways they find most fulfilling.[68] Requiring people to spend money on ozone reductions reduces the funds they have available for other things they need and desire, such as housing, education, food, health care, leisure, and transportation, as well as for other safety-enhancing measures.[69] Most of these things have a direct or indirect effect on health, and all of them affect overall welfare and quality of life. In other words, the costs imposed by regulatory measures such as ozone-reduction requirements make people worse off. Environmental regulations are, therefore, not pure health-improvement measures, but rather impose unavoidable tradeoffs among a range of competing goals.

CARB's ozone standards will make Californians better off overall only if health benefits of lower ozone exceed the harm to health and welfare caused by the costs of reducing ozone. Making such a demonstration is all the more crucial because most of the costs of air-pollution regulations are hidden. Thus, the people who are ostensibly being helped by lower ozone levels are never made aware of the real tradeoffs they've made and therefore have no way to determine whether they've struck a good bargain. CARB did not weigh these tradeoffs or even acknowledge their existence when adopting its ozone standards. I show here that because the costs of reducing ozone are large and the benefits small, requiring attainment of CARB's standard will make Californians worse off overall.

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[68] A. P. Bartel and L. G. Thomas, "Predation through Regulation: The Wage and Profit Effects of the Occupational Safety and Health Administration and the Environmental Protection Agency," *Journal of Law and Economics* 30 (1987): 239; D. Schoenbrod, "Protecting the Environment in the Spirit of the Common Law," in *The Common Law and the Environment: Rethinking the Statutory Basis for Modern Environmental Law*, ed. R. E. Meiners and A. P. Morriss (Lanham, Md.: Rowman & Littlefield, 2000); A. Wildavsky, *Searching for Safety* (New Brunswick, N.J.: Transaction Publishers, 1988).

[69] The costs of environmental regulations are also regressive, falling more heavily on the poorest. See F. B. Cross, "When Environmental Regulations Kill: The Role of Health/Health Analysis," *Ecology Law Quarterly* 22 (1995): 729, and H. D. Robinson, "Who Pays for Industrial Pollution Abatement?" *Review of Economics and Statistics* 67 (1985): 702–6.

The Net Effects of CARB's Ozone Standards Should Have Been Evaluated Prior to Their Adoption

CARB asserts on p. 1-6 of its staff report that "the proposed ambient air quality standards will in and of themselves have no environmental or economic impacts. Standards simply define clean air." CARB puts forward this argument to excuse its decision not to provide any analysis of the costs of attaining its proposed ozone standards. On the other hand, CARB provides a detailed estimate of the predicted health benefits of attaining its standards. But by CARB's own logic, just as a standard imposes no costs, a standard confers no benefits. Only the act of attempting to attain the standard can impose costs or confer benefits.

By omitting discussion of costs while discussing benefits in detail, CARB has created the false impression that the predicted health benefits of its ozone standards can somehow be delivered without imposing any offsetting hardships on the people who are ostensibly being helped by the tougher standards. CARB's standards will unavoidably do both and will therefore have potentially profound impacts on Californians' prosperity.

CARB claims the full effects of trying to attain its ozone standards will be weighed when it comes time to adopt the regulations necessary to attain the standards.[70] This might seem reasonable outside of the real-world context of air-pollution regulation. What harm could it do to adopt a standard, so long as we know that we'll address the tough questions before we actually start trying to attain it?

Unfortunately, the net health and welfare effects of CARB's standards will never actually be addressed. Now that the new eight-hour standard has been adopted, regulators and environmental activists will ensure that the standard becomes its own justification. The new standard is now the official government-sanctioned delineation between "safe" and "unsafe" air. Because the new standard is more stringent, it will be exceeded much more frequently—about two to three times more frequently—than the current federal eight-hour standard, creating more ozone-alert days and bigger, scarier numbers of "bad-air" days in regulators' and activists' reports and press releases. The result will be greater public fear for any given level of ozone and greater pressure for bold action to alleviate the alleged crisis.

The act of setting a pollution standard is intended to create a strong rhetorical justification for doing whatever is necessary to attain it. Regulators and activists will point to California's failure to attain the standard as an urgent and serious threat to public health that must be remedied, and will work to augment the authority of state and local regulators to impose the new regulations and requirements necessary to attain the standard. The unacceptable alternative, they will say, will be to leave people in danger.

[70] California Air Resources Board, *Review of the California Ambient Air Quality Standard for Ozone*, p. 1-6.

By delaying an evaluation of the net effects of the proposed standard until *after* it was adopted, CARB preempted the scrutiny that would have determined whether the standard was worth adopting in the first place. Now that it has been adopted, the question of whether it is worth attaining will no longer be in play. Rather, the political debate will center on *how* to attain it. The new standard will work like a ratchet, permanently moving policy debates and regulatory activities to a new regime. CARB has thus assumed that which remains to be demonstrated—that attaining its new standard is worthwhile, regardless of how much people will have to give up to get there; that among all the opportunities available for improving welfare, reducing ozone below the current federal eight-hour standard is the best use of people's scarce resources; and that the asserted benefits of the additional ozone reductions are relatively certain to materialize.

Another factor that will work against the promised accounting of costs and benefits is that the standard is now enshrined in California law. It is true that CARB does not have legal authority to mandate attainment of its ozone standards. However, the standards should not be seen as merely symbolic. CARB has broad regulatory authority over many sources of air pollution, including motor vehicles, and can invoke this authority in the service of attaining its stricter standards.

As shown by previous efforts to attain other air-pollution standards, discussions of how to attain a standard that *must* be attained are not based on analysis of net health and welfare benefits.[71] Rather, federal, state, and local regulators have developed over the years customary norms for what is a cost-effective air-pollution control measure. These norms are based on political limits on the burdens regulators can impose on given industries and consumers, rather than on whether incurring the costs is worth it, given the benefits that will be obtained from incurring them, or on whether a given measure is the most cost-effective means available to obtain a given amount of pollution reduction. Setting a pollution standard thus has the effect of placing attainment of that standard ahead of other public and private goals and priorities. Once again, the standard will become its own justification.

In this light, CARB's successful attempt to separate the setting of a new standard from the unavoidable effects of attempting to attain that standard can be seen as a shrewd political strategy to get the standard adopted with a minimum of scrutiny, knowing that such scrutiny will be of little consequence now that the standard is safely in place. By claiming that its ozone standards have no ill effects on Californians' overall welfare, CARB has swept under the rug exactly the issues that should determine whether reducing

[71] The EPA adopted the federal eight-hour ozone standard in 1996, despite the agency's own conclusion that the measures necessary to attain it would impose social costs twice as great as the value of the benefits of the ozone reductions. See Environmental Protection Agency, *Regulatory Impact Analyses for the Particulate Matter and Ozone National Ambient Air Quality Standards and Proposed Regional Haze Rule* (Washington, D.C., July 17, 1997), www.epa.gov/ttn/oarpg/naaqsfin/ria.html.. Outside analysts believe the real costs of attaining the EPA's ozone standard will be several times greater than EPA predicted. For a review of these estimates, see R. Lutter, "Head in the Clouds Decisionmaking: EPA's Air Quality Standards for Ozone," in *Painting the White House Green: Rationalizing Environmental Policy inside the Executive Office of the President*, ed. R. Lutter and J. F. Shogren (Washington, D.C.: Resources for the Future, 2004).

ozone to a given level is worthwhile. The health benefits of attaining CARB's standards and the hardships imposed by the actions necessary to attain them are inextricably linked.

The Relationship between Wealth and Health

Higher incomes are associated with lower health risks, because people spend a portion of each additional dollar of income on things that directly or indirectly improve health and safety, such as better health care, more crashworthy cars, and more nutritious food.[72] Higher incomes are also associated with better health habits, such as decreased smoking and drinking, and increased exercise.[73] Regulations reduce people's effective incomes by redirecting some expenditures toward paying the costs of the regulations.[74] As a result, regulatory costs worsen health.

To put it another way, a regulation might reduce mortality by reducing ozone, but the costs of the regulation also increase mortality by diverting expenditures away from other health- and safety-enhancing activities. This is known as a "risk-risk" or "health-health" tradeoff. If the regulatory costs are large enough, the regulation will cause net harm, killing more people than it saves.

Based on these tradeoffs, researchers estimate that every $17 million in regulatory costs induces one statistical death.[75] Taking account of the risk-risk tradeoffs due to the costs of ozone reductions thus puts a strong constraint on how much we should be willing to pay to achieve the benefits CARB predicts from reducing ozone. CARB predicts the incremental benefit of its standard would be to reduce mortality by three hundred lives per year. But attaining the standard will kill an additional three hundred people per year if attaining the standard costs $5.1 billion per year. I showed in the previous section that attaining CARB's standard is likely to save far fewer than three hundred lives. Accounting for publication bias would reduce by about 50 percent CARB's estimate of lives saved by ozone reductions. Counting only reductions in ozone only down to the level of CARB's standard would reduce the number of lives saved by 80 percent. Accounting for model uncertainty would reduce the estimated number of lives saved by ozone reductions to zero.

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[72] R. Lutter, J. Morrall III, and W. Viscusi, "The Cost-Per-Life-Saved Cutoff for Safety-Enhancing Regulations," *Economic Inquiry* 37 (1999): 599–608; W. K. Viscusi, "The Value of Risks to Life and Health," *Journal of Economic Literature* 31 (1993): 1912–46; Wildavsky, *Searching for Safety*.

[73] W. K. Viscusi, "Mortality Effects of Regulatory Costs and Policy Evaluation Criteria," *The Rand Journal of Economics* 25 (1994): 94–109.

[74] R. Keeney, "Estimating Fatalities Induced by the Economic Costs of Regulations," *Journal of Risk and Uncertainty* 14 (1997): 5–23; R. Keeney and K. Green, *Estimating Fatalities Induced by Economic Impacts of EPA's Ozone and Particulate Standards* (Los Angeles: Reason Public Policy Institute, June 1997), http://www.rppi.org/environment/ps225.html; Viscusi, "Mortality Effects of Regulatory Costs and Policy Evaluation Criteria";" Wildavsky, *Searching for Safety*.

[75] Lutter, Morrall, and Viscusi, "The Cost-Per-Life-Saved Cutoff for Safety-Enhancing Regulations." The value is adjusted from 1997 to 2004 dollars based on the Consumer Price Index (CPI).

Attempting to attain CARB's standard will cost much more than $5 billion per year, and will therefore kill many more people than it saves. The South Coast Air Quality Management District (SCAQMD), estimates that attaining just the current federal one-hour ozone standard in South Coast will cost about $4 billion per year in 2010, and $6 billion in 2020.[76] This is likely an underestimate of the real costs, because 70 percent of the emission reductions needed to reach one-hour attainment are so-called "black box" measures that have yet to be identified.[77] For these measures, SCAQMD assumed the cost per ton of emission reductions would be the same as the average cost for the measures already identified. These are just the costs in South Coast. Several other areas of the state also violate the one-hour ozone standard and will incur additional costs to attain it. The incremental cost of going from attainment of the federal one-hour standard to attainment of the federal eight-hour standard will be even larger. A recent study estimated this cost at $16.6 billion per year in South Coast.[78] Costs for attaining the current eight-hour standard statewide will be higher still.

Taken together, these estimates suggest that it would cost more than $20 billion per year for South Coast to go from current ozone levels down to attainment of the federal eight-hour standard. Statewide costs would add billions to this figure. Incremental costs for attaining CARB's eight-hour standard would be higher still. Everyone in the state lives in an air basin where at least one monitoring location violates CARB's standard, usually by a large margin. Furthermore, the marginal cost per ton for reducing ozone-forming emissions will be higher for CARB's eight-hour standard than for the less-stringent federal standards, because the most cost-effective emission-reduction opportunities will already have been used to attain the less-stringent standards. CARB's standard also requires a larger incremental reduction in eight-hour ozone levels than the current federal eight-hour standard. Going from attainment of the federal one-hour ozone standard to attainment of the federal eight-hour standard requires roughly a 0.010 ppm reduction in peak eight-hour ozone levels. But going from the federal eight-hour standard to CARB's standard would require at least a 0.025 ppm reduction in peak eight-hour

[76] SCAQMD estimates the costs in 1997 dollars to be $3.5 billion in 2010 and $5 billion in 2020. I've adjusted the costs to 2004 dollars. Some of these costs could be attributed to PM reductions, since reductions in ozone-forming pollutants also reduce PM. However, even if half the costs of the NOx and VOC reductions are attributed to PM, the cost of ozone reductions are still far in excess of the benefits.

[77] South Coast Air Quality Management District, *2003 Air Quality Management Plan, Appendix III: Base and Future Year Emission Inventories* (Diamond Bar, Calif., February 2003), http://www.aqmd.gov/aqmp/AQMD03AQMP.htm.

[78] R. Lutter, *Is EPA's Ozone Standard Feasible?* (Washington, D.C.: AEI-Brookings Joint Center for Regulatory Studies, December 1999), www.aei.brookings.org/publications/reganalyses/reg_analysis_99_06.pdf. This analysis included an assumption that pollution-control costs would decrease by 5 percent per year, due to technological advancement. Dollar values were reported as 1990 dollars, and I have adjusted them to 2004 dollars based on the CPI. This cost estimate assumed all measures to attain the standard would be implemented by 2010.

ozone levels.[79] Attempting to attain CARB's standard is thus likely to impose tens of billions per year in incremental costs on California's citizens.

The large costs of attaining CARB's standard will cause a large net reduction in people's health and welfare. For example, assuming an incremental cost of $20 billion per year for statewide attainment of CARB's standard—about $1,700 per California household—the standard would, on net, cause more than eight hundred additional premature deaths each year. The real cost of attaining CARB's eight-hour ozone standard is likely to be much greater than $20 billion per year.

It isn't even clear that attaining the current federal eight-hour ozone standard is practical in South Coast or a number of other parts of California, including much of the San Joaquin Valley. Recent modeling studies suggest that attaining the federal eight-hour standard would require reductions in nitrogen oxides (NOx) and/or volatile organic compounds (VOC) of 80 to 90 percent below 1999 human-caused emissions levels in these areas.[80] These large emission reductions are necessary because eight-hour ozone levels have proved less sensitive to ozone-precursor reductions than one-hour peak levels.

Full attainment of CARB's eight-hour standard is likely to be impossible in many areas, regardless of cost. Even with a complete elimination of all transportation, industrial, farming, and solvent emissions, which would in itself be a miraculous feat, there would still be some natural emissions of VOC and NOx from vegetation and soil microbes.[81] These emissions account for about 20 percent of total VOC emissions and probably a few percent of NOx emissions.[82] Tires, upholstery, and carpeting emit small amounts of VOC as well, while fires create some NOx. Even human exhalations contain small amounts of VOC and nitric oxide (NO; a component of NOx), amounting in the case of VOC to as much as perhaps 0.5–1.0 percent of the emissions inventory in an

[79] The federal eight-hour standard is set at 0.085 ppm, while CARB's standard is 0.070 ppm. However, the federal standard is based on the fourth-highest daily ozone reading. Thus, even under attainment of the federal eight-hour ozone standard, peak ozone levels would be higher than 0.085 ppm (see notes to figure 3).

[80] See, for example, S. Reynolds, C. L. Blanchard, and S. D. Ziman, "Understanding the Effectiveness of Precursor Reductions in Lowering 8-Hr Ozone Concentrations," *Journal of the Air & Waste Management Association* 53 (2003): 195–205.

[81] A. Guenther, C. Geron, T. Pierce, et al., "Natural Emissions of Non-Methane Volatile Organic Compounds, Carbon Monoxide, and Oxides of Nitrogen from North America," *Atmospheric Environment* 34 (2000): 2205–30; L. C. Marr, D. R. Black and R. A. Harley, "Formation of Photochemical Air Pollution in Central California. 1. Development of a Revised Motor Vehicle Emission Inventory," *Journal of Geophysical Research* 107 (2002): 5-1–5-9; P. Solomon, E. Cowling, G. Hidy, et al., "Comparison of Scientific Findings from Major Ozone Field Studies in North America and Europe," *Atmospheric Environment* 34 (2000): 1885–1920; R. L. Tanner and B. Zielinska, "Determination of the Biogenic Emission Rates of Species Contributing to VOC in the San Joaquin Valley of California," *Atmospheric Environment* 28 (1994): 1113–20.

[82] Marr, Black, and Harley, "Formation of Photochemical Air Pollution in Central California. 1. Development of a Revised Motor Vehicle Emission Inventory."

urbanized area, or several tons per day in an area as populous as South Coast.[83] Gas stoves, ovens, and other appliances powered by fossil fuels emit NOx, and SCAQMD attributes about 2 percent of 2003 NOx emissions to residential fuel combustion.[84] Ozone and ozone-forming emissions are transported from outside California as well, so some of California's ozone—as much as 0.05 ppm on some days—comes from elsewhere.[85]

Even if all industrial, commercial, and transportation emissions could somehow be eliminated without massive hardship, ozone-prone areas such as San Bernardino, Riverside, Bakersfield, and Fresno would likely find it impossible to attain CARB's standard, due to remaining incidental emissions from human activity and transport from outside. Natural emissions plus outside transport might be sufficient to cause a violation of CARB's standard in these areas. This suggests that CARB's standard is impossible to attain and incompatible with human habitation in several areas of the state.

Health Benefits from Ozone?

Ozone up in the stratospheric ozone layer protects us from the sun's ultraviolet (UV) rays, which can cause skin cancer and cataracts at high enough exposures. Ozone near ground level, including human-caused ozone, adds to this protection. Indeed, even without human-caused ozone, about 10 percent of the total ozone above our heads is found within ten miles of sea level.[86]

The EPA performed an internal analysis in 1997 estimating that the incremental ozone reductions (beyond the federal one-hour ozone standard) necessary to attain the federal eight-hour standard would result in an additional seven hundred cases of nonmelanoma skin cancer each year nationwide, due to increased exposure to solar UV light. The EPA never officially made this analysis public.[87] Estimates by the Department

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[83] See, for example, J. D. Fenske and S. E. Paulson, "Human Breath Emissions of VOCs," *Journal of the Air and Waste Management Association* 49 (1999): 594–98; F. L. Ricciardolo, P. J. Sterk, B. Gaston, et al., "Nitric Oxide in Health and Disease of the Respiratory System," *Physiological Reviews* 84 (2004): 731–65. Fenske and Paulson estimate that human VOC exhalations might make up from a few tenths of a percent to a few percent of the total emissions of various VOCs in an urbanized area. Given this estimate, human exhalations probably contribute a few tons per day of the approximately one thousand tons of VOC per day in the SCAQMD's estimated VOC inventory for the late 1990s.

[84] South Coast Air Quality Management District, *2003 Air Quality Management Plan, Appendix III: Base and Future Year Emission Inventories*.

[85] T. Watson, "Air Pollution from Other Countries Drifts into USA; Emissions That Cross Borders Could Cancel out U.S. Efforts," *USA Today*, March 14, 2005. Also see http://www.psat.wa.gov/Publications/03_proceedings/PAPERS/ORAL/7b_schwa.pdf, http://www.epa.gov/airtrends/international.html, and http://atmos.chem.le.ac.uk/group/psm_group_reprints/04_acpd-4-transport.pdf,

[86] United Nations Environment Program, *Scientific Assessment of Ozone Depletion: 2002* (Nairobi, Kenya, 2002), http://www.unep.org/ozone/sap2002.shtml.

[87] The EPA analysis is now posted at aei.brookings.org/admin/pdffiles/php9v.pdf. It suggests that average summer ozone levels would need to be reduced by from one to a few ppb in most eight-hour nonattainment

of Energy (DOE) suggest that reducing ozone to the eight-hour standard would also result in a few thousand additional cases of cataracts, a few dozen cases of melanoma skin cancer, and several melanoma deaths each year.[88]

In order to reach full attainment of CARB's eight-hour standard, most populated areas of the state would have to reduce typical daily eight-hour ozone levels during the ozone season by about four to eight times the amount assumed in the EPA analysis cited above. The number of Californians living in regions that violate CARB's standard is about one-fourth the number of people nationwide who live in regions that violate the current federal eight-hour standard. Thus, as a very rough estimate, we might expect that the incremental harm to Californians from attaining CARB's standard is at least as large as the national estimates from the EPA and DOE.[89] If so, we would expect attainment of CARB's standard to cause several hundred cases of nonmelanoma skin cancer, a few thousand additional cases of cataracts, and several melanoma deaths each year. Whatever the actual harm from reduced shielding of solar UV light due to ground-level ozone reductions, CARB should have performed an estimate of these effects and weighed them against the potential health benefits of its standard.[90]

Even after receiving public comments highlighting the negative effects of lower ozone levels, CARB's staff continued to avoid the issue. At CARB's April 28, 2005 governing board hearing to adopt its proposed ozone standards, CARB's staff stated that it believed "it is likely that any such effect would be very small because the change in UVB absorption would be restricted to only a very short path length, typically a few hundred meters. The limited literature on this topic does not support the commenter's contention."

CARB's staff did not cite any of the research literature and provided no actual calculations to support its claims. CARB's staff also failed to mention to the governing board that back in the late 1990s, both the EPA and the DOE had found the UV health effects of lower ground-level ozone to be certain enough to provide quantitative estimates of increases in skin cancers and cataracts.

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areas in order to attain the standard. Also see R. Lutter and H. Gruenspect, "Assessing Benefits of Ground Level Ozone: What Role for Science in Setting National Ambient Air Quality Standards?" *Tulane Environmental Law Journal* 15 (2001): 85–96.

[88] Department of Energy, *EPA Docket A-95-54, IV-D-2694, Appendix B-9* (Washington, DC: March 21, 1995), cited in Lutter and Gruenspect, "Assessing Benefits of Ground Level Ozone." The DOE estimates were actually higher than this, but DOE assumed a 0.01 ppm reduction in seasonal average ozone levels. Smaller reductions would be necessary to attain the eight-hour standard in most areas, and I have adjusted the estimates downward to reflect this.

[89] Four times the ozone reduction multiplied by one-fourth the number of people.

[90] A federal appeals court ordered the EPA to include the offsetting harm from lower ozone in assessing the health benefits of its eight-hour ozone standard, though the agency failed to abide by the court's requirement. See http://www.epa.gov/airlinks/uvb-fs.pdf, and Lutter and Gruenspect, "Assessing Benefits of Ground Level Ozone"

A number of research papers have indeed assessed the relationship between total atmospheric ozone and the amount of solar UV light reaching the ground, and the relationship between UV exposure and health damage. Based on this research, scientists with the United Nations Environment Program (UNEP) estimate that each 1 percent decrease in total atmospheric ozone results in a 1–2 percent increase in human exposure to biologically active UV light—that is, wavelengths of light associated with particular health effects or genetic damage.[91] The UNEP estimates that a 10 percent increase in UV exposure would result in one to two hundred new skin cancers per year per million people.[92] Thus, in a population the size of California, a 1 percent increase in UV exposure would be expected to result in about 350 to 700 new cases of skin cancer each year.[93] Such an increase in UV light reaching the ground would occur if total ozone above ground level declined by 0.5–1.0 percent. The ozone reductions necessary to attain CARB's new eight-hour standard could easily be of this magnitude.[94]

The evidence suggests that reducing ground-level ozone by amounts necessary to attain CARB's eight-hour ozone standard is likely to result in hundreds of new cases of skin cancer and thousands of new cases of cataracts each year. The direct harm to public health from lower ozone levels is thus large enough to offset much of the health benefit CARB claims for lower ozone levels. Indeed, if low-level ozone does not kill people, as argued above, then the extra cancers caused by increased solar UV exposure would likely more than offset all of the other health benefits CARB claims for ozone reductions.

CARB claims that the effect of any increase in UV exposure would be "very small." Yet even by CARB's own estimates, the health effects of ozone exposure are also very small. CARB also does not have similar reservations about regulating other very small risks. For example, CARB considers a cancer risk from airborne toxic compounds to be unacceptable if the predicted risk is greater than one in one million.[95] But the ozone reductions necessary to attain CARB's eight-hour ozone standard could easily cause an increase in cancer risk of ten in one million, or ten times CARB's threshold level for regulating other cancer risks. Given that CARB eagerly highlights and regulates much

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[91] United Nations Environment Program, *Environmental Effects of Ozone Depletion: 1998 Assessment* (Nairobi, Kenya, 1998), http://www.gcrio.org/UNEP1998/UNEP98.html. The chapters of this report were also published as separate papers in the October 1998 issue of *Journal of Photochemistry and Photobiology B*, available at http://www.gcrio.org/ozone/toc.html.

[92] Ibid.

[93] Based on ten to twenty skin cancers each year per million people for a 1 percent increase in UV exposure and a population of 35 million.

[94] The total amount of ozone from the ground to the top of the atmosphere is roughly three hundred Dobson Units. If daily summertime ozone were reduced by, say, 30 ppb up to an altitude of two miles, this would reduce total ozone by one and a half Dobson Units, or 0.5 percent.

[95] It should also be noted that regulatory cancer-risk estimates include assumptions that guarantee a substantial overestimate of the true risk. Thus, CARB regulates cancer risks that are in reality orders of magnitude lower than one in one million. See, for example, B. N. Ames and L. S. Gold, "The Causes and Prevention of Cancer: Gaining Perspective," *Environmental Health Perspectives* 105 (1997): 865–74.

smaller and less certain risks in other circumstances, it seems safe to conclude that the agency has ignored the risk of UV exposure from lower ground-level ozone not because of a lack of scientific support, but because of the political and bureaucratic inconvenience of acknowledging the issue.

Cost-Benefit Analysis

The risk-risk analysis presented above shows by itself that attempting to attain CARB's standard will do far more harm than good to California's citizens. A more comprehensive method of assessing regulatory programs is cost-benefit analysis (CBA). A CBA compares the estimated value of all costs and benefits expected to arise due to implementation of a regulatory program to determine whether that program will result in net benefits to societal welfare.

Many people find CBA distasteful or even morally repugnant, because of the suggestion that a value can be placed on human lives. But we are all natural cost-benefit analysts, deciding, for example, whether to pay more for a car with side-impact airbags or antilock brakes, whether to install state-of-the-art sprinkler systems in our homes or incur a greater risk of injury by choosing a house with stairs, whether to take a riskier job that pays a higher salary, or even whether to risk driving to a restaurant rather than eating at home. Many people who could afford them nevertheless choose not to purchase sprinkler systems or more-crashworthy cars, and many people are willing to incur a greater risk of injury or death in return for a higher salary or a more spacious multilevel home. When we make these choices we implicitly place values on our lives, even if we would prefer not to think of it in those terms. Reducing ozone imposes similar tradeoffs, and cost-benefit analysis is an essential way to assess whether the tradeoff is a good one.

A number of studies have estimated the implicit values people place on their lives based on the actual risk-benefit decisions they make.[96] These studies suggest that, on average, people value their own lives at around $7 million. Other studies have similarly placed values on avoiding various health impacts, such as an emergency room visit or a hospitalization.[97] For example, EPA scientists recently published a study estimating the national benefits of attaining the federal eight-hour ozone standard.[98] This study assumed, for instance, a value of $6.5 million per premature death, $18,000 per hospital admission, and $75 per school absence.

[96] For a recent review see W. K. Viscusi, "The Value of Life: Estimates with Risks by Occupation and Industry," *Economic Inquiry* 42 (2004): 29–48.

[97] See, for example, estimates cited in Hubbell, Hallberg, McCubbin, et al., "Health-Related Benefits of Attaining the 8-hr Ozone Standard."

[98] Ibid.

Based on $7 million per premature death avoided, the numbers used by EPA for other health effects,[99] and CARB's estimates of various health effects avoided, the benefits of attaining the federal eight-hour ozone standard in California would total $2.6 billion per year. The incremental benefits of CARB's standard would total $2.3 billion per year. Ninety percent of these benefits are due to avoiding premature deaths. These benefits are only a small fraction of the likely costs of meeting the respective standards. If ozone is not causing premature death, then the costs of attempting to attain CARB's standard would exceed the benefits by more than a factor of one hundred. These estimates do not account for the negative health effects of lower ozone levels. If lower ozone causes several hundred new cases per year of skin cancer and several thousand new cases of cataracts, this alone would offset much of its direct health benefit.

When CARB adopted its new eight-hour ozone standard, it also reaffirmed its preexisting one-hour standard. CARB's one-hour standard is somewhat more stringent than the federal eight-hour standard, but substantially less stringent than CARB's eight-hour standard. Because the incremental costs of ozone reductions are far larger than the incremental benefits, attempting to attain this standard would likewise make Californians worse off overall.

It should not be surprising that the costs of reducing ozone beyond the federal eight-hour standard far outweigh the benefits, because the EPA has already concluded that attaining even the less stringent federal eight-hour standard would do more harm than good. When the EPA adopted the federal eight-hour ozone standard in 1997, the agency had already concluded that the incremental costs of attaining the standard would outweigh the incremental benefits by about a factor of two.[100] Outside analysts estimated that the actual ratio of costs to benefits would be more than a factor of ten, because the EPA made implausible assumptions that lowered the projected attainment costs.[101] It only makes sense that CARB's standard would have an even less favorable cost-benefit ratio, because the marginal cost of each increment of ozone reduction would continue to increase as emissions limits were progressively tightened.

Involuntary vs. Voluntary Risks

One might argue that air pollution is different from other risks, because the risk is involuntary. This brings up the question of whether air pollution violates people's right to be free from unreasonable harms caused by others. But even when a risk is involuntary, we still have to ask at what level it becomes unreasonable. People who plant flowers, shrubs, and grass in their yards cause emissions of additional pollen and mold spores that

[99] EPA used a value of $6.5 million per death, which is lower than the number I've used here. My $7 million figure is based on Viscusi, "The Value of Life."

[100] Environmental Protection Agency, *Regulatory Impact Analyses for the Particulate Matter and Ozone National Ambient Air Quality Standards and Proposed Regional Haze Rule.*

[101] Lutter, "Head in the Clouds Decisionmaking."

can aggravate other people's allergies or asthma. When people drive, they increase the risk that someone else might be killed in a car accident.

For example, epidemiological studies of airborne outdoor pollen and mold spores often report risks of serious health effects, such as emergency room visits and hospitalizations, and minor health effects, such as coughing, of a similar magnitude to air pollution.[102] While we might think of pollen as "natural" in the sense of not being caused by humans, in arid California most pollen in urbanized areas comes from people planting lawns, plants, shrubs, and trees on their property.

The national-average risk of dying in a car accident is about 1 in 7,000 per year—many times higher and far more certain than the ostensible risk of death from ozone. One might argue that people voluntarily bear the risk of driving, but most people would probably consider as involuntary the risk of getting hit by a drunk driver, a red-light-runner, a tailgater, or a driver who took his eyes off the road while reaching for a music CD. Pedestrians might also consider their risk of being killed by a motor vehicle to be borne involuntarily.

According to CARB's estimates, failing to attain its ozone standard would result in an additional 1-in-120,000 risk of dying each year, an additional 1-in-18,000 risk of ending up in the hospital due to respiratory distress, and, among children with asthma, an additional 1-in-4,000 risk of going to the emergency room. These pollution risks seem very small in an absolute sense, and also compared to other involuntary risks people face, suggesting that protection from them shouldn't necessarily be considered a legal or moral right.

The degree to which a risk is involuntary is itself ambiguous. For example, the population of the San Bernardino-Riverside area doubled between 1960 and 1980, and doubled again between 1980 and 2000. Yet this area had the worst air pollution in the country even *before* those people moved there. Furthermore, publicity over the area's high smog levels, as well as its manifestly poor visibility, put the area's high pollution levels in the category of "common knowledge." To be sure, virtually everyone would prefer less air pollution to more. Nevertheless, like people everywhere else, the millions of people who decided to live in the San Bernardino-Riverside region voluntarily chose an inseparable "package" of qualities, some pleasant, some neutral, and some unpleasant, which taken together added up to a desirable place to live.

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[102] See, for example, M. B. Lierl and R. W. Hornung, "Relationship of Outdoor Air Quality to Pediatric Asthma Exacerbations," *Annals of Allergy, Asthma, and Immunology* 90 (2003): 28–33; L. M. Neas, D. W. Dockery, H. Burge, et al., "Fungus Spores, Air Pollutants, and Other Determinants of Peak Expiratory Flow Rate in Children," *American Journal of Epidemiology* 143 (1996): 797–807; D. M. Stieb, R. C. Beveridge, J. R. Brook, et al., "Air Pollution, Aeroallergens and Cardiorespiratory Emergency Department Visits in Saint John, Canada," *Journal of Exposure Analysis and Environmental Epidemiology* 10 (2000): 461–77; and A. Tobias, I. Galan, J. R. Banegas, et al., "Short Term Effects of Airborne Pollen Concentrations on Asthma Epidemic," *Thorax* 58 (2003): 708–10. Of course, the same concerns apply to these studies as for epidemiological studies of air pollution health effects. For a brief review, see R. W. Atkinson and D. P. Strachan, "Role of Outdoor Aeroallergens in Asthma Exacerbations: Epidemiological Evidence," *Thorax* 59 (2004): 277–78.

Another way to assess the cost-benefit picture is to transfer the costs and benefits to more familiar ground and ask whether most people would find the costs reasonable. For example, about 1,600 motor-vehicle occupants are killed in crashes each year in California.[103] Reducing this number by 300, the estimated incremental benefit of CARB's standard, would require a 19 percent improvement in the safety of automobiles. Californians purchase about two million automobiles per year. If we assume that the incremental cost of attaining CARB's standard is a good deal at $20 billion per year, this is analogous to making the implausible assumption that most people would be willing to pay an extra $10,000 for a car that reduces their annual risk of death in an auto accident by 19 percent. We face hundreds of risks every day, both large and small. We would impoverish ourselves and end up far less safe and healthy if we tried to buy such expensive risk reductions in every aspect, or even a few aspects, of our lives.

Reducing Ozone is a Poor Risk-Reduction Option

Reducing ozone is only one among hundreds or thousands of ways to improve people's health and welfare. While we would all choose to save three hundred people's lives if we could, what if there were other things we could do that would save a thousand lives or more for the same investment? Surely we would choose to save a thousand rather than only three hundred.

Even assuming a low incremental cost of $20 billion per year to attain CARB's standard, ozone reductions would then cost somewhere between about $1.7 million and $800 million per year of life saved. The lower figure assumes an average gain of forty years of life per person for the three hundred premature deaths prevented by ozone reductions. This would be the case if ozone were killing mainly people in their prime. The higher cost-figure assumes an average gain of one month of life per person, which would be closer to the case if ozone were killing mainly people who were already sick and who would have died soon in any case. Of course, the ozone reductions might on average delay death by even less than a month or not at all.

Based on an assessment of more than five hundred lifesaving measures, researchers at the Harvard School of Public Health concluded that the median lifesaving measure costs about $55,000 per year of life saved—far less than the cost of ozone reductions.[104] In other words, even assuming an unrealistically low cost for attaining CARB's standard, choosing merely the median risk-reduction measure would add between thirty and fourteen thousand times more years to people's lives when compared with reducing ozone.

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[103] Data for 2002 available at http://www.applications.dhs.ca.gov/epicdata/scripts/broker.exe?_SERVICE=Pool2&_PROGRAM=programs.cause_age.sas&REGION0=XXX&R1=F+2002&REGION=California&OUTPUT=HTML. This number excludes 1,300 deaths where the role of the person who was killed (occupant or pedestrian, for example) was unspecified.

[104] T. O. Tengs, M. E. Adams, J. S. Pliskin, et al., "Five-Hundred Life-Saving Interventions and Their Cost-Effectiveness," *Risk Analysis* 15 (1995): 369–90. Tengs et al. estimated the cost at $42,000 in 1993 dollars. I've adjusted the value in the text to 2004 dollars.

We could glibly say that we should undertake all available risk-reduction measures and save as many lives as possible. But this begs the question. If we lived in a world of infinite resources and omniscience about the full consequences of our actions, then we would, of course, undertake literally all health and safety measures available. But in such a world there would be no politics or policy debates over environmental regulations or anything else. Politics and policy debates exist exactly because resources and knowledge are scarce and insufficient to satisfy all our needs and aspirations. We have to make tradeoffs among a range of competing needs and desires. Maximizing human welfare requires spending these scarce resources in ways that maximize improvements per dollar invested.

One might argue that talking about other ways to reduce risk is irrelevant, because it is not as if money is sitting around waiting to be spent on risk reductions, with ozone just one of many choices. We can choose to reduce ozone or not, but if we choose not to, this does not mean the government will fund some other risk-reduction measure(s). The flaw in this line of reasoning is that it implicitly assumes that only publicly determined risk-reduction priorities and expenditures are legitimate. In fact, if Californians aren't forced to spend money to attain CARB's ozone standards, they will have more money to spend as they see fit. People will spend these funds in various ways, some of which will directly or indirectly improve health, welfare, and quality of life. People will spend their money in whatever ways they find most useful and rewarding, given their particular values, needs, and aspirations. As a result, they will be better off than if they had been forced to spend it on ozone reductions that delivered tiny benefits compared to the costs imposed.

This doesn't excuse California's elected officials and regulators from their duty to prioritize tax revenues in ways that generate the maximum risk reduction per dollar invested. Through idealistic legislation such as CARB's mandate to keep reducing ozone until not the slightest chance of harm can be detected, even in an unrealistic worst-case analysis, California's elected officials have ensured that Californians spend large sums of money to achieve tiny benefits. Whatever amount of money California's elected officials choose to devote to risk reduction, these funds should be prioritized so as to deliver maximum benefits per dollar invested.

Conclusion

CARB's ozone standard will impose great hardship while conferring few benefits. Even by CARB's own estimates, reducing ozone from current levels will result in small and imperceptible improvements in public health. The expected benefits are even smaller after we account for CARB's overestimate of the likely health benefits from additional ozone reductions. On the other hand, attempting to attain CARB's ozone standard will impose costs on Californians likely to be in the range of tens of billions of dollars per year. Indeed, CARB's standard is probably impossible to attain in much of the state. Reducing ozone will also increase Californians' exposure to harmful solar UV light.

The question therefore arises as to why CARB would seek to impose an ozone standard that would harm the people the agency intends to protect. CARB's goal is to provide everyone with clean, safe air—something we all agree is crucial. But as a powerful, single-purpose agency with a staff that is passionate about air quality, CARB unavoidably suffers from tunnel vision—the pursuit of a single-minded goal to the point where it does more harm than good.[105]

We have many needs and aspirations, but limited resources of money, time, knowledge, and attention. This forces us to make implicit and explicit tradeoffs every day based on our goals, tastes, circumstances, and financial means. But CARB deals only in air-pollution reduction, and places its mission ahead of other people's particular desires. Despite already stringent standards, CARB will pursue the next increment of air-pollution reduction, and the next, regardless of whether the increasingly marginal benefits are worth having or the costs worth bearing, given all the other things that people will have to give up in the bargain. By pursuing marginal and uncertain health benefits at great cost, CARB will make Californians worse off overall.

[105] S. Breyer, *Breaking the Vicious Circle: Toward Effective Risk Regulation* (Cambridge, Mass.: Harvard University Press, 1993).

Appendix III

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

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

By Joel Schwartz

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

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

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

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

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

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

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

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False Alarm on Asthma and Air Pollution

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

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

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

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

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

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

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

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

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

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

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

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

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

Much Ado about Very Little

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

Monkey Business

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

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

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

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

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

Of Mice and Men

By far the most serious health claim about air pollution is that it kills tens of thousands of Americans each year, mainly due to exposure to $PM_{2.5}$. There is no question that high levels of air pollution can kill. About 4,000 Londoners died during the infamous five-day "London Fog" of December 1952, when soot and sulfur dioxide soared to levels tens of times greater than the highest levels experienced in developed countries today, and visibility dropped to less than 20 feet.[34]

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

Sins of Omission

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

The Politics of Air Pollution Health Science

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

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

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

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

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

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

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

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

Notes

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

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

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

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

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

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

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

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

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

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

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

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