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11-26-14

Environmental Protection Agency EPA Docket
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1200 Pennsylvania Avenue, NW. Washington,
DC 20460.

Office of Management and Budget
Office of Information and Regulatory Affairs Attn: Desk
Officer for the EPA
25 17th St. NW.
Washington, DC 20503

Ms. Amy Vasu, Sector Policies and Programs Division
(D205–01), U.S. EPA, Research

Public Comments on EPA Proposed Power Plant Rule
Docket ID No. EPA-HQ-OAR–2013-0602

Email submission—per web access

Ladies and Gentlemen:

My comments on the Clean Power Plan, Docket reference above, are based on my long time objections to the science that EPA uses to justify its adoption of policy and promulgate regulations under the Clean Air Act.

In the submission I will outline my objections to EPA conduct, specifically:

1. EPA misuse of epidemiological studies and small associations to suggest toxic effects of air pollutants to justify regulations of small particles and ozone and claim those regulations produce human health benefits.
2. EPA misconduct in matters related to compliance with the Information Quality Act and avoidance of conflicts of interest by EPA funded researchers.
3. Failure of the EPA to properly vet the IPCC assessment reports and claims of the IPCC about warming and the negative impacts of warming.

In my submission I will also show that:

1. Warming, if it does occur, will improve quality of life.
2. The co-benefits claimed by the EPA from small particle and ozone reductions are the product of unreliable junk epidemiology.
3. The socioeconomic harm caused by the economic burdens imposed by new regs should not be ignored and are significant.

The EPA builds its case for the CPP rules on speculation about warming and its negative effect, based on unjustified reliance on IPCC modeling already proven to be inaccurate. The projected harm of warming must be evaluated in the real world of evidence that warm is healthier for humans and the planet. My position is that if any warming does occur, it would be beneficial to human health, not detrimental. My position on the claim of the EPA that carbon dioxide will cause warming is that nothing of a sort has occurred in spite of an increase of carbon dioxide, a trace gas of 20 Parts per Million or about 0.002% increase in the ambient air.

In the initial roll out of the CPP proposal the EPA emphasized co- benefits from small particle and ozone reductions by means of their epidemiological studies. They ignored or de-emphasized in their promotions the lack of evidence to prove the presence of and the dangers of warming. I assert that their claims of deaths and asthma attacks prevented are based on bad science. I also have research in this submission that shows no human harm from ambient level of small particles and ozone. .

I will detail the proven health benefits of warming, if it would occur, in the submission attached.

I will also include a paper on socioeconomic deprivation as a cause of reductions in life expectancy and human welfare. Increasing the cost of living by these regulations will kill people prematurely and cause economic deprivation.

I thank you for your consideration of my submission and trust that responsible people will consider the consequences of findings and proposed solutions and hold to the Clean Air Act language that obligates the EPA to find, identify and verify scientifically reliable evidence of greenhouse gas and air pollution effects and determine if there is a human and environmental harm that must be mitigated.

Sincerely,

/s/ JDunn MD

John Dale Dunn MD JD

Comments on EPA's Proposed Power Plant Rule
EPA Docket ID No. OAR–2013-0602

Introduction

The EPA failed to establish the valid Science that supports it's claims about the need for and benefits of its Clean Power Plant rules.

The EPA assumes that by pounding the table it can establish that the weak and tenuous Endangerment Findings are adequate under the Clean Air Act (CAA) to establish a scientific foundation that establishes a harm and thus the required statutory basis for a regulatory regime such as the proposed Clean Power Plant Rules (CPP). However the proper conduct of the agency on establishing the requisite danger or toxic effect from warming is still not adequate. The CCA is clear that eht EPA must develop reliable science on assertions of air pollution that harms and then propose proper mitigation of that harm. This submission will show a failure by the EPA and its acceptance of trust me science and speculation by the IPCC.

The EPA suggests that the concentrations of GHGs in the atmosphere may reasonably be anticipated to endanger public health and welfare of current and future generations, but that is only on the basis of second hand information, accepted as true from sources that clearly have a political agenda. That is not the kind of scientific inquiry required in the Clean Air act.

It appears the EPA had no intention of doing its own research and vetting the IPCC claims for veracity and reproducibility. However there is a problem with how when the information is modeling already proven incorrect and speculation on effects of warming that have not occurred. That is not the basis for good policy making under the CAA.

EPA has, thus far, failed to establish that it was proceeding with anything other than a wink and nod, by accepting IPCC assessment reports, even if they contain failed modeling and nothing more than assertions without proof of the harm to occur because of the unproven but projected warming. There is no evidence of accurate modeling, and there is no evidence of harm. There is only a theory.

In fact the mechanism for harm is lost in the fact that the IPCC assumed warming would occur and it has not.

My disbelief and the disbelief of many others that study the work of this latest agency regulatory plan is the result of a lack of compliance with proper verification and validation procedures known to scientists and requires appropriate actionand scientific validation by the EPA as it studies its own work and the work of its reference organizations and researchers.

The public relations crusade of the EPA that attempts to dress up the Clean Power Plant Rules as a benefit to human welfare is unique in that it jumps right to the co-benefits of reducing small particles and ozone, which obligates me to focus on that malpractice in epidemiology.

EPA Misuse of inadequate epidemiological research

My objections to the scientific misconduct of EPA sponsored researchers are based on accepted methods and rules for epidemiology and toxicology outlined by nationally known experts writing in the Federal Judicial Center's book, now in its 3rd edition, *Reference Manual on Scientific Evidence.*

The *Reference Manual* is intended to educate federal judges, hearing officers and magistrates on admissibility of scientific testimony and evidence and is provided to all federal judges for their working libraries (it is also available digitally to anyone for free, *(PDF is available from The National Academies Press at http://www.nap.edu/catalog.php?record_id=13163)*.

For example in the epidemiology chapter at page 549, three prominent authors, one attorney and two epidemiologists, Green, Freedman, and Gordis, warn researchers to reject small associations as proof of anything because of the problems of false positive and negative errors due to confounders, and adhere to the rules that robust associations must be present in observational uncontrolled studies to support a claim of proof of causation.

My position is that the mistaken use of small associations as proof of causation riddles all the EPA research on air pollution human health effects. Data dredging comparing deaths to air pollution monitor information, aggravated by the EPA researcher misconduct in calling premature deaths any deaths that occur on days when the death rate is more than the average, leads to small associations of pseudo premature deaths, leads to claims of small effects of deaths, leads to projections to the population of the country that justify a picture of a health crisis that doesn't exist at all. Woops, then the Exec of the EPA testifies they have proof of hundreds of thousands of deaths from ambient air pollution that doesn't kill anyone anytime anywhere in America.

From the *Reference Manual* page 599

> The factors that guide epidemiologists in making judgments about

causation

(and there is no threshold number that must exist) are144

1. Temporal relationship,

2. Strength of the association,

3. Dose–response relationship,

4. Replication of the findings,

5. Biological plausibility (coherence with existing knowledge),

6. Consideration of alternative explanations,

7. Cessation of exposure,

8. Specificity of the association, and

9. Consistency with other knowledge.

There is no formula or algorithm that can be used to assess whether a causal inference is appropriate based on these guidelines.145 One or more factors may be absent even when a true causal relationship exists.146 Similarly, the existence of some factors does not ensure that a causal relationship exists. Drawing causal inferences after finding an association and considering these factors requires judg- ment and searching analysis, based on biology, of why a factor or factors may be absent despite a causal relationship, and vice versa. Although the drawing of causal inferences is informed by scientific expertise, it is not a determination that is made by using an objective or algorithmic methodology.

These guidelines reflect criteria proposed by the U.S. Surgeon General in 1964 in assessing the relationship between smoking and lung cancer and expanded upon by Sir Austin Bradford Hill in 1965 (148) and are often referred to as the Hill criteria or Hill factors.

At page 601

B. How Strong Is the Association Between the Exposure and Disease?

The relative risk is one of the cornerstones for causal inferences.[156] Relative risk measures the strength of the association. The higher the relative risk, the greater the likelihood that the relationship is causal.[157] For cigarette smoking, for example, the estimated relative risk for lung cancer is very high, about 10.[158] That is, the risk of lung cancer in smokers is approximately 10 times the risk in nonsmokers.

A relative risk of 10, as seen with smoking and lung cancer, is so high that it is extremely difficult to imagine any bias or confounding factor that might account for it. The higher the relative risk, the stronger the association

> and the lower the chance that the effect is spurious. Although lower relative risks can reflect causality, the epidemiologist will scrutinize such associations more closely because there is a greater chance that they are the result of uncontrolled con- founding or biases.

EPA officials like Lisa Jackson should be ashamed that they make stupid claims about air pollution like her claim that air pollution was as serious as cancer, as she did in a Congressional Hearing, when she asserted that small particle air pollution was the most dangerous toxin on earth. EPA was exposing people to this most dangerous toxin in labs across the country, so who's lying, the experimenters or the EPA officials?

http://epahumantesting.com/the-most-toxic-substance-on-earth/.

The EPA by custom and practice, sponsors and funds, then accepts and references research that violates the rule on strength of association (relative risk), respected throughout all of the scientific community except in air pollution studies and toxicology studies sponsored by the EPA, where ends justify the means. The EPA compounds that scientific misconduct by redefining or allowing their researchers to redefine premature deaths to something other than death before life expectancy—nothing more than death on the day when rates are higher. For example, in the EPA junk science project air pollution research, a 99 year old man who dies a cardio respiratory death on a day that the death rate is higher than the average and a day when the lag time from elevated air pollution is right—is a premature death due to air pollution. There is not even the modicum of effort to properly define premature, or even to establish the cause of the death.

That is, in a simple medical analysis, redefining premature death and death from a toxic effect. No point in discussing the silliness of ambient air pollution being blamed for a death that could easily be attributed to other causes.

To explain the scientific principles for establishing causation is relatively simple as taken from the text of the Reference Manual on Scientific Evidence (3rd ed. 2011, National Academies Press), but it is necessary to review the federal jurisprudence on admissibility of scientific testimony and evidence—pertinent to administrative and civil proceedings.

Legal decisions on scientific evidence

The courts and administrative law judges have been decidedly inadequate in applying admissibility standards for the proposed science to support EPA regulations under the CCA, regularly deferring to agency discretion and refusing to apply rules on arbitrariness and capriciousness under the rules of the Administrative Procedure Act. The courts that defer to the agency on all matters of science fail to read and apply the guidance of *Daubert v Merrill Dow, 509 US 579 (1993)* as well as its progeny *Joiner* (1997) and *Kumho Tire* (1999) and the very direct and instructive guidance provided by the *Reference Manual On Scientific Evidence* (3rd ed. National Academies Press, 2011) sponsored and supervised by the Federal Judicial Center.

There are clearly Congressional and administrative processes that could stop scientific misconduct. The legal, statutory or administrative validity of the EPA assertions on endangerment are not final at all, anymore than the previous policy positions and underlying science used for air pollution regulations under the admonitions, provisions and clear directives of the Clean Air Act. The EPA doesn't get to do it once and then be done with it; they must continue to evaluate the claims made. The policies adopted and the impact of regulatory decisions. A regulatory regime must be modified and amended based on the most reliable science available.

After more than 2 decades of unreliable epidemiology sponsored by the EPA on small particle air pollution, a better analysis of the EPA research and a reassessment of the claims of hundreds of thousands of deaths from small particles and asthma from ozone would be quite salutary.

Confessions of human experiment misconduct by EPA officials

2 years ago a lawsuit was filed attempting to stop EPA air pollutant human exposure experiments that were being conducted at the University Of North Carolina School Of Medicine in an EPA laboratory under the Institutional Review Board supervision of UNC.

The lawsuit was filed in Virginia Federal District Court, styled as

American Traditions Institute v. US EPA in Federal District Court Alexandria Division Eastern District Virginia. Civil Action No. 1:12-CV-1066-AJT-TCB.

In the preliminary discovery period documents produced by both sides included Declarations under penalty of perjury to be submitted to the District Court by the parties.

Declarations by the plaintiff parties focused on the fact that human exposure experiments were unethical when the EPAs executive declared that small particle air pollution was toxic and lethal.

US EPA executive Lisa Jackson declared in testimony before congress in 2010 the EPA position on small particle air pollution, that it was toxic, lethal and killed hundreds of thousands of Americans every year:

http://epahumantesting.com/the-most-toxic-substance-on-earth/

EPA officials responsible for the human experiments at UNC made these revelations in their declarations under penalty of perjury:

Wayne Cascio MD declared that 10 medical schools in the United States and 6 foreign schools were involved in human exposure experiments using air pollutants.
http://junksciencecom.files.wordpress.com/2013/12/declaration-cascio-highlighted.doc

Martin Case PhD, supervisor of the research activities, declared that he told subjects they might die from the experiments, but did not obtain written consent with that warning. Such a consent would still be unethical, since international accords prohibit human experiements that present a risk of death with very limited and extraordinary exceptions.

http://junkscience.com/2012/10/05/epa-admits-to-court-human-subjects-may-die-from-air-pollution-experiments/ http://junksciencecom.files.wordpress.com/2013/12/declaration-case-highlighted.doc

Robert Devlin PhD, chief research supervisor for the EPA human exposure experimental program, declared that the human experiments had been conducted for many years and were necessary because epidemiological studies were not reliable proof of air pollution harm.

http://junksciencecom.files.wordpress.com/2013/12/declaration-devlin-highlighted.doc

The important part of Dr. Devlin's declaration begins at paragraph 6:

Epidemiological studies typically use data from large populations of people with varying susceptibility to PM2.5 and evaluate the relationship between short or long-term changes in ambient levels ofPM2.5, e.g. changes in the 24-hour average level ofPM2.5 measured at monitors in a metropolitan area, with changes in mortality and morbidity such as the numbers of emergency department visits and hospital admissions. This generally involves the use of complex statistical methods to evaluate the mathematical relationship between variations in measured ambient air pollution levels and health data.

7. Epidemiological observations are the primary tool in the discovery of risks to public health such as that presented by ambient PM2.5. However, epidemiological studies do not generally provide direct evidence of causation. They indicate the existence or lack of a statistical relationship between ambient levels of PM2.5 and adverse health outcomes. Large population studies cannot assess the biological mechanisms (called biological plausibility) that could explain how inhaling ambient air pollution particles can cause illness or death in susceptible individuals. This sometimes leaves open the question of whether the observed association in the epidemiological study is causal or whether PM2.5 is merely a marker for some other unknown substance.

8. Controlled human exposure studies conducted by EPA scientists and EPA funded scientists at multiple universities in the United States fill an information gap that cannot be filled by large

population studies. In 1998 the Committee on Research Priorities for Airborne Particulate Matter was established by the National Research Council in response to a request from Congress. The committee was charged with producing four reports over a five-year period which describe a conceptual framework for an integrated national program of particulate-matter research and identified the most critical research needs linked to key policy-related scientific uncertainties. Excerpts from their most recent report (published in 2004) are attached as Exhibit

1 to this Declaration. On page 36 the Committee says:

Controlled human exposure studies offer the opportunity to study small numbers of human subjects under carefully controlled exposure conditions and gain valuable insights into both the relative deposition of inhaled particles and the resulting health effects. Individuals studied can range from healthy people to individuals with cardiac or respiratory diseases of varying degrees of severity. In all cases, the specific protocols defining the subjects, the exposure conditions, and the evaluation procedures must be reviewed and approved by institutional review boards providing oversight for human experimentation. The exposure atmospheres studied vary, ranging from well-defined, single-component aerosols (such as black carbon or sulfuric acid) to atmospheres produced by recently developed particle concentrators, which concentrate the particles present in ambient air. The concentrations of particles studied are limited by ethical considerations and by concern for the range of concentrations, from the experimental setting to typical ambient concentration, over which findings need to be extrapolated. <u>End of excerpt</u>.

How can the EPA propose to use the epidemiological studies with small associations going on into the future and expect the public to accept the assertions, when the EPA's own scientists say the studies are not reliable? This epidemiological misconduct is the basis for the EPA claim of human benefits from the Clean Power Plant Rules?

The EPA doesn't even bother to speculate on the weak assertions that relate to greenhouse gas effects and the harm that result from the predicted warming?

Congress, representing the interests of the public, must deny the EPA credibility on its claims of air pollution dangers until the EPA has comprehensively and publicly demonstrated that the peer review processes it had employed to validate the "major assessments" supporting the Administrator's CAA Section 202(a)(1) Findings satisfied the letter *and* spirit of U.S. law.

However that is not enough—the past claims of the EPA on air pollution dangers are clearly not reliable and are subject to a thorough review immediately and a serious vetting of the epidemiology that is promoted not only by the EPA but also its' Clean Air Scientific Advisory Committee (CASAC), which includes individuals that do the same kind of research

for the EPA, put up the same kind of studies Dr. Devlin says don't prove anything. Millions of dollars have exchanged hands when the PhDs traded their status and credentials for money so the EPA could make unreliable and specious claims about air pollution dangers.

An example of the exaggerations and unscientific claims even from top advisors of the EPA on Scientific issues is this claim by Jon Samet, MD MPH, Chair of the CASAC for more than a decade. Dr. Samet advises an agency involved in human exposure experiments approved by the CASAC at the same time takes money for research from EPA and has friends and colleagues and is affiliated with institutions that take EPA grant money. Not surprisingly, Dr. Samet now is pushing a no threshold, linear model for air pollutants, and says there is no safe level of air pollution. He made his comments in an editorial in one of the most prestigious medical journals in America:

http://www.nejm.org/doi/full/10.1056/NEJMp1103332

There is no limit to the gall of the EPA clean air freaks—they don't' understand that the Clean Air Act is actually, as written, the SAFE AIR ACT. No natural air can ever be "clean" as the EPA or its leadership and researchers want it, but no threshold toxicology allows arbitrary ratcheting down of air standards with no end in sight. In fact the sensitivity of the monitors seems to be the driving force, and, for example Parts per billion ozone standards continue to be reduced to what now are close to or below natural ambient levels. Where do we go from the air quality of the real world? Ozone and small particles are produced by nature. Can we remove dust and ozone from the real world, and for what purpose except a regulatory fetish?

EPA Has Failed to do Proper Science Reviews

I defer to the submission of and in depth commentary by Lawrence Kogan of the Institute for Trade Standards and Sustainability Development (ITSSD), that probes the requirements under the Information Quality Act, also called the Data Quality Act.

In a submission under this docket on the CPP dated August 13, 2014, author Kogan details a lengthy exchange between ITSSD and the EPA on FOIA requests and the Institute's continuing concern about the empty EPA assertions that it has fulfilled its obligations to do due diligence with regards to its reliance on the IPCC Assessment Reports that it relies on to assert the effects of carbon dioxide and greenhouse gasses in its Assessment Reports as well as the harm that will come from any warming. I cannot disagree with Kogan that the EPA is exposed as a mendacious and negligent agency, willing to proceed without any effort to act in accordance with the law. Shame on them.

To date it appears that EPA has not produced records validating the IQA compliance of other agencies' transmittal memoranda and peer review reports containing author

responses to peer reviewer, agency and public comments. EPA points only to the brief *pro forma* certifications of IQA.

Mr. Kogan pointed out that at least twenty-five commenters complained about the IPCC third and fourth assessment reports and there is no evidence that the EPA did anything other than accept them without a proper vetting as required by law.

That certainly makes things easier for the EPA, but it leaves the question unanswered—how is it that a federal agency takes the work of a political entity with an agenda and assumes that the work product is reliable? I know that the IPCC and the EPA have a common goal of controlling and reducing industrial activity and activities that produce carbon dioxide, but does that justify scientific and policy misconduct under the statutory guidelines of the CAA?

Is it reasonable for the EPA to claim that that regulations that reduce small particle or ozone levels will improve quality of life or reduce harm? Only if the evidence supports the claim. Of course there is no evidence about warming, because warming has not occurred, and models have been failures on predicting warming. Predictions on the harm of warming are speculation and ignore the benefits of warming. Since the EPA operates under the provisions of the law, the Clean Air Act and laws that pertain to integrity of scientific inquiry, it must show a serious effort to identify causes of harm and then suggest proper mitigation.

I am not comforted to know that the EPA asserts that the IPCC's information quality process is consistent with EPA's *Guidelines for Ensuring and Maximizing the Quality, Objectivity, Utility and Integrity of Information Disseminated by the Environmental Protection Agency.*"

Mr. Kogan also points out in his submission that the EPA still needs to disclose specific records that would reveal satisfactory level 3 and 4 IQA compliance. More and better is in the original in depth submission from ITSSD here:

https://nebula.wsimg.com/9293ff84df35eecadd25e73a03499114?AccessKeyId=39A2DC689E4CA87C906D&disposition=0&alloworigin=1

Mr. Kogan points out conflicts of interest, reviewers who reviewed for the EPA what they had done for the IPCC, for example, and multiple failures to comply with IQA guidance on implementation and failures to comply with interagency peer review rules as well as failures to identify sources of uncertainty in climate impacts and models. The Modeling failures have now become a climate studies joke, but the EPA doesn't evaluate the modeling and the repeated failures? For what are their rules about scientific integrity if not to check methodology and accuracy, as well as reliability and veracity, of work and results. Short term verification of modeling predictions is basic stuff and the EPA doesn't bother? Maybe because it has a dog in the fight for modeling being correct and would prefer looking the other way.

In his discussion of these **three basic complaints about EPA compliance** Mr. Kogan points

out that "In response to the latter two sets of comments, **EPA referred to "the same likelihood and probability terminology assigned to climate science findings by the IPCC and USGCRP/CCSP**."[50] In addition, **it referred stakeholders to the ranges of uncertainty in the "assumptions about future concentrations of GHGs and aerosols in the various scenarios considered by the IPCC and the differing climate sensitivities of the various climate models used in the simulations.** EPA rationalized that its reference to the USGCRP/CCSP and IPCC reports upon which the Administrator's CAA Section 202(a)(1) Findings had relied had been transparent and would enable the reproducibility of such information by third parties.

However, EPA has yet to disclose records revealing how it had validated the reproducibility of the assumptions, theories and extrapolations underlying the computer models and datasets supporting such HISAs."

Mr. Kogan also asserts that the EPA Cannot Rely on the IPCC's 2013–2014 Fifth Assessment Report (AR5) or the USGCRP's 2014 "Climate Change Impacts in the United States" (Third National Climate Assessment ("NCA3-2014) as the Scientific Foundation For Its Proposed Power Plant Rule, Since EPA & DOC-NOAA Failed to Validate Such Science in Conformance With the Information Quality Act (44 U.S.C. 3516 note).

I agree with the analysis and conclusions of Mr. Kogan on conflicts of interest that prevent objective review of scientific research. His evidence is compelling but not surprising considering the billions spent to support universities and environmental interest groups.

Mr. Kogan says, and I agree

> "Several conclusions may be drawn with respect to these findings. At the very least, these findings demonstrate that DOC-NOAA (among other federal agencies) is significantly financially and policy- "invested" in the climate science research of forty-three U.S. universities, nonprofit institutes and activist ENGOs, which have returned in-kind via their scientists' substantive contributions to IPCC AR5 and/or NCA3-2014. This serves several purposes. First, such research drives scientific, legal and political support for EPA's proposed new existing power plants GHG emissions rule which, together with the President's Climate Change Data initiative,[81] helps implement, on a domestic level, the President's Climate Change Action Plan.[82] Second, it helps to ensure the White House will be taken more seriously at international climate change negotiations.[83] Third, such research more broadly drives international political support for the President's Global Climate Change Initiative intended to persuade developing countries to undertake local actions consistent with this regulatory direction and international efforts to secure agreement on a post-Kyoto Protocol GHG multilateral emissions control treaty."

Warming, if it does occur, will benefit the biosphere

I am familiar with and agree with the assertions supported by evidence from around the globe that warming, if it does occur, would be salutary for the biosphere and humankind. That evidence is well displayed in the 1000 pages with thousands of references, in the book *Climate Change Reconsidered II. Biological Impacts*, one of a series on Climate, this book edited by Drs. Fred Singer, Sherwood Idso, Criag Idso, and Robert Carter, at:

http://heartland.org/sites/default/files/full-report-ccr-ii-biological-impacts.pdf

Chapters on other parts of the biosphere are 1-6, the outline of the human effects chapter and summary of conclusions is here:

Sure, I would like the EPA to start following the guidance of the law on scientific relevance and I think there has been entirely too little oversight by the congress or tests of scientific validity by congress or the courts, but cleaning up the EPA junk science mess must start somewhere and this is another opportunity to make some chicken soup. I assert that the EPA doesn't have a dangerous gas but a political agenda supported weakly by assertions that have no scientifically reliable basis.

Carbon Dioxide is essential to life, not toxic, and invisible, tasteless and odorless so it can't even get into the EPA regulatory regime on aesthetics, but the EPA calls the new crisis Carbon Pollution to follow its old methods. The projections that increases in CO2 will cause warming are not working out so well but, as a physician, I assert that a warming of the planet can clearly be shown to be beneficial to living things. The current average temperature of Planet Earth is in the high 50s Fahrenheit, still too cold for a naked human and is associated with a limited growing season at the higher latitudes. Warming with increase food production and milder nighttime and winter climate can be shown to reduce premature deaths and illness.

Wintertime is a killer in the high latitudes and summertime is not a killer at the low latitudes as I will demonstrate in this submission. Death effects from extreme warm spells are caused by poor acclimation and housing and involve harvesting effects of people already in bad health. On the other hand winter and cold spell deaths are premature and do not show a harvesting effect—in fact the cold spells show a tale of deaths that continues out beyond the cold spell, due to infectious and vascular impacts. The research that shows cold increases in infectious diseases, vector diseases, and vascular diseases (heart attacks and strokes) are convincing and can't be refuted.

The compilation of these realities on the benefits of warm versus cold for the plant and animal life, the environment and human welfare, around the globe, are at chapter 7 of the 1000 page Text of the Series *Climate Change Reconsidered II.* part of the series, *Biological Impacts*, edited by Drs. Fred Singer, Sherwood and Craig Idso, and Robert Carter, at:

http://heartland.org/sites/default/files/full-report-ccr-ii-biological-impacts.pdf

Chapter 7

Human Health
Key Findings

Introduction

7.1 Hot vs. Cold Weather
- 7.1.1 Asia
- 7.1.2 Europe
- 7.1.3 North America
- 7.1.4 Other Regions
- 7.1.5 Multiple Regions

7.2 Cardiovascular Disease

7.3 Respiratory Disease

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7.6 Dengue Fever

7.7 Tick-Borne Diseases

7.8 Diet
- 7.8.1 Antioxidants
- 7.8.2 Common Food Plants
- 7.8.3 Medicinal Plants
- 7.8.4 Health-Harming Substances

Key Findings

The following bulleted points summarize the main findings of this chapter:

Warmer temperatures lead to a decrease in temperature-related mortality, including deaths associated with cardiovascular disease, respiratory disease, and strokes. The evidence of this benefit comes from research conducted in every major country of the world.

In the United States the average person who died because of cold temperature exposure lost in excess of 10 years of potential life, whereas the average person who died because of hot temperature exposure likely lost no more than a few days or weeks of life.

Some 4,600 deaths are delayed each year as people in the U.S. move from cold northeastern states to warm southwestern states. Between 3 and 7% of the gains in longevity experienced by the U.S. population over the past three decades is due simply to people moving to warmer states.

Cold-related deaths are far more numerous than heat-related deaths in the United States, Europe, and almost all countries outside the tropics. Coronary and cerebral thrombosis account for about half of all cold-related mortality.

Global warming is reducing the incidence of cardiovascular diseases related to low temperatures and wintry weather by a much greater degree than it increases the incidence of cardiovascular diseases associated with high temperatures and summer heat waves.

The adverse health impacts of cold temperatures, especially with respect to respiratory health, are more significant than those of high temperatures in many parts of the world, including Spain, Canada, Shanghai, and Taiwan. In the subtropical island of Taiwan, for example, researchers found low minimum temperatures were the strongest risk factor associated with outpatient visits for respiratory diseases.

A vast body of scientific examination and research contradict the claim that malaria will expand across the globe and intensify as a result of CO_2-induced warming.

Concerns over large increases in vector-borne diseases such as dengue as a result of rising temperatures are unfounded and unsupported by the scientific literature, as climatic indices are poor predictors for dengue disease.

While climatic factors largely determine the geographical distribution of ticks, temperature and climate change are not among the significant factors determining the incidence of tick-borne diseases.

The ongoing rise in the air's CO_2 content is not only raising the productivity of Earth's common food plants but also significantly increasing the quantity and potency of the many health-promoting substances found in their tissues, which are the ultimate sources of sustenance for essentially all animals and humans.

Atmospheric CO_2 enrichment positively impacts the production of numerous health-promoting substances found in medicinal or "health food" plants, and this phenomenon may have contributed to the increase in human life span that has occurred over the past century or so.

There appears to be little reason to expect any significant CO_2-induced increases in human-health-harming substances produced by plants as the atmosphere's CO_2 concentration continues to rise.

Co-benefits from the CPP rules are silly

I have made my case above about the co-benefits claimed by the EPA and the weak associations that the EPA relies on to claim benefits are risible.

The last 25 years of EPA research on air pollution have all shown their dependence on fallible and weak claims about air pollution harmful effects. Contained here in this submission are research papers that show no such thing.

And, to paraphrase Einstein, a single experiment performed by a researchers can disprove the most elegant and attractive theory. **The EPA has what it thinks is an attractive theory about the multifarious effects of air pollution, but what if that theory is nothing but ridiculous claims? What if the research by Entrom and Milloy shows that the claims about small particulates and ozone are not supported by good science?.**

There are consequences to mindless and unjustified EPA rules on regulation of economic, industrial and energy activities.

In appendix G below Steenland explains the harmful effects of socioeconomic deprivations and depredatons of the the EPA Clean Power Plant Rules—that will cause energy costs to rise, impacting the lower economic strata of the society in a negative way.

It is essential that the EPA reorient itself to serving the populace at large, rather than a select group of individuals motivated by environmental issues that produce excess regulation of economic activities that cause hardship.

Time for the EPA to start acting like it cares about the poor and the downtrodden and tailoring its regulatory actions to avoid hardships for the poor.

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Appendix A

Small particle air pollution Submission to EPA by John Dale Dunn MD JD

I submit here a previous submission I made to EPA on Small Particle air pollution critical of the human epidemiological research on small particle air pollution. Criticizing proposed new standards on small particles as not justified.

The EPA violations of epidemiological and toxicology scientific rules are a scandal that cannot be ignored. The Dockery 1993, Pope 1995, and Samet 2000 studies (see endnotes) and other studies of health effects of air pollution relied on by the EPA, all showed that large studies with adequate power could not demonstrate relative risk of any significance. The studies all showed effects less than ten percent, rather than the statistically and scientifically required 200 to 300 percent effect. It is astounding the EPA has the gall to announce an air pollution crisis and propose more stringent air quality standards when none of the studies the EPA relies on show and proof of health effects.

The EPA is obligated to educate the public on the clear evidence that air pollution may have aesthetic and cultural import, but that there is no air pollution health "crisis." The EPA and its sponsored and supported health effects researchers are now just raising their voice in this debate instead of trying to use science. The EPA air pollution health effects science is an emperor with no clothes, as discussed below.

This commentary challenges the EPA to show one study that proves that one person has died due to air pollution in America in this past 20 years. People die for various reasons, suddenly and not so suddenly, as will be discussed below. That reality eludes the work of numbers crunchers who slave at desks over death certificate information like Pope and Dockery. One doesn't die from an exposure to air pollution, one dies from failed medical therapy, arrhythmias caused by long term coronary disease, stroke, pulmonary embolism, which are not caused by air pollution. The Asthma problem is an increasing problem not related to air pollution, since the rate of asthma is increasing with decreasing air pollution. The deaths from asthma will be discussed below and have nothing to do with air pollution, it is a socioeconomic phenomenon. It is time to retire the air pollution health effects studies of crude death tallies and its time for the EPA to stand down from this repeated use of crisis talk and aggressive pursuit of pure air—a religious campaign disguised as science in the public interest.

As a last and compelling consideration, this author is familiar with death in America. As an emergency physician, much more familiar with what kills people than economists and public health officials who don't know which is the business end of a ventilator and live in the world of death certificates and mortality data. People die for many reasons and under many circumstances in America, but air pollution doesn't kill them, even the worst levels of outdoor air pollution one might imagine in America don't create a toxic level, which reveals the other major flaw in the EPA crisis rhetoric, junk science toxicology that completely disregards any effort to define toxin or toxicity. That subject will also be dealt with herein below.

The scientific epidemiological and toxicological criticisms of the EPA health effects studies and policy making are:

1. The Dockery 1993 and Pope 1995 studies did not show valid evidence of death effects, since they showed a death effects relative risk below 1.1, a negligible relative risk that is 10 percent of the minimal relative risk all epidemiologists consider necessary for proof of causation. A 200% or 300% change in death effect is the lower limit. Some epidemiologists require relative risk of 4 or a 400% effect when evaluating poorly controlled cohort studies.
2. This relative risk problem cannot be overcome by EPA and health effects researchers emphasizing the misleading use of the term statistical significance, which is not a proof test, but a statistical reliability test. One can be statistically confident and reliable but absolutely wrong.
3. The EPA and its health effects researchers have consistently and persistently ignored the lack of proof of health effects in these studies, and have made public announcements and allowed media reports to proclaim that thousands are dying in America due to air pollution when the studies do not show any proof of death effect at all. Lying for justice or an environmental ideal does not make the lie any less dishonest.
4. The health effects research used by the EPA has consistently ignored the basic rules for toxicology and the well-known phenomenon of threshold for toxicity. Only at the EPA does straight line toxicology have any status, mostly because it avoids serious science. Main stream toxicology science is still committed to the idea of threshold of effect and the old saying—the dose makes the toxin. The EPA scientists in house know the truth, but again politics and a commitment to a policy/environmental ideal results in lies.
5. Under no valid scientific analysis retro or prospectively, can the EPA use the methodologies or the results of the Pope, Dockery, McDonnell, or Lipfert (see endnotes) studies to justify one more burdensome air pollution regulation, but there is strong evidence for rescinding the last round of Air Quality Standards.
6. The EPA has a mandate to act only on the basis of acceptable scientific evidence of health effects, and is obligated to abandon the precautionary principle approach to regulatory policy, a pathetic substitute for legitimate science and clearly a principle founded in politics, not science.
7. The EPA could never convince a Federal Court, operating under Federal Rules of Evidence 702 and the court dicta for expert and scientific testimony that the EPA air pollution health effects science is valid proof of anything. The Pope, Dockery and Lipfert or Samet studies cannot be massaged or misrepresented enough to create any proof of air pollution health effects. The studies show trends within an insignificant range and "associations," that are not evidence of proof of health effects.
8. Precautionary principles that are used by the EPA as stand-alone policy justification are nothing more than a dressed up version of anxiety, cannot pass muster for admissible scientific evidence in a Federal Court and ignore the reality of risk/benefit analysis.
9. Based on the information reviewed in this critique, the EPA must revisit old regs, forgo new, more onerous and expensive regulatory interventions, and the EPA must suspend its rule making in air pollution until it can find valid and reliable science on health effects.

Toxic air pollution existed in the past, and still may occasionally occur in some places on the planet as a local phenomenon, as particulate and other noxious air pollution in industrial areas, from various sources. Certainly air in big cities, Pittsburg, Los Angeles, Houston, New York was fouled in the past by air pollutants and even when not toxic, was smelly and visible, but trends in air pollution in the past 30 years as reported and confirmed by the EPA, have all been positive, attributable to changes in industrial processes, regulatory efforts and cleaner petroleum and

coal consumption. Any study or discussion of air pollution is focused on a moving, improving problem. However the public thinks the air is worse than ever and there is an air pollution health effects crisis, and that is the fault of the EPA, its favorite researchers, and the mass media, who love to scare the public, since EPA budgets and environmental organization budgets depend on the anxiety of the public.

The death and illness rates during smog and air pollution catastrophe periods in the past were affected by less effective medical management and heavier cigarette smoking but also significantly higher air pollution than exists anywhere in the United States today, for many reasons. Deaths from acute respiratory failure in the past were more common and less preventable, but that is an independent factor related to medical advances and not due to air pollution itself. Airway diseases, the main effect of any air pollution, were less treatable before the 1970s. Pulmonary Medicine has changed dramatically for the better since 1970. Many airway diseases were more dangerous in the past and medical therapies frequently failed to control disease and death. Medical expertise in respiratory illness and cardiovascular disease is changed, but Pope and Dockery still yearn for the good old days of killer air because it scares the public. Their research ignores the trends of the last 20 years and below I will discuss a conscious deception in the second half of the Pope research from NCI data. In addition the EPA air pollution researchers continue to ignore the weakness of their findings, hoping to keep alive the "deadly air" panic talk.

People die for lots of reasons in America, but not due to air pollution. Air pollution health effects researchers know that, but act as though nothing has changed. The EPA should carefully reevaluate the number of deaths that researchers claim are due to air pollution in the last 20 years, but the EPA has a conflict of interest. No air pollution crisis might mean reduced EPA funding. No air pollution crisis might mean no funding for the researchers and their support organizations.

The air pollution health effects studies are based on weak epidemiologic relationships and trends carelessly described without definition as "associations," or "trends." Well ice cream consumption and drowning or boating accidents are associated by season, but ice cream eating doesn't cause water accidents. Associations are not proof, they are observations of phenomena--clusters of events that may or may not mean something. Epidemiologists know these things and should be careful when describing data associations and trends within insignificant ranges like less than relative risk of 2, so that the reader or reporter won't mislead the public or a politician. However, the definitions are not forthcoming from the scientists and researchers because saying that there is no crisis of air pollution means no publications for air pollution researchers, no invitations to swell events, no funding, no chance to pursue a political agenda and change the world, making your mother proud.

The uncertainties of the air pollution health effects studies, the weak relative risks and the methodological problems of the most influential of the health effects studies are so noticeable and remarkable that during this comment period the EPA should reassess what has gone wrong in air pollution health effects research. The EPA should assess how these weak studies have affected EPA policy and rule making. The EPA doesn't have the right to panic the public and political leaders with deceptive junk science in the service of religious and fanatic environmentalism.

DISCUSSION OF THE STATISTICAL AND METHODOLOGIC PROBLEMS OF THE SAMET, POPE, AND DOCKERY HEALTH EFFECT DEATH STUDIES.

Author's comments are in bold. Studies referenced are underlined and the cite is in the endnotes by name and year. Sorry to disappoint those who want numbered endnotes—not a formal paper.

J. Samet (Samet 2000) published in the *New England Journal of Medicine,* a study modeled after the studies of Pope (1995) and Dockery (1993). He compiled and studied deaths in twenty American cities over a period of years, and compared them with air pollution monitor reports for those cities.

Samet in this 2000 paper asserts the following:

--"the relative rate of death from all causes was 0.51 percent increase for each increase in the PM 10 (10 micron size particulates) of 10 micrograms per cubic meter." **This effect is not proof of anything, and Dr. Samet knows it. Less than a 1-% death effect is a nonsense result in a big cohort study.**

--"the relative rate of death from cardiovascular and respiratory diseases rises 0.68 percent for each increase of 10 micrograms per cubic meter" Trends **of less than 1% inside of a meaningless range of relative risk less than 1.05? A serious epidemiologist would snicker?**

--"we also analyzed the effects of levels of carbon monoxide, sulfur dioxide, and nitrogen dioxide in a fashion similar to that of the analysis of pm 10 levels. After adjustment for pm 10 and ozone levels we found little evidence that these pollutants had a significant effect on the relative rate of death." **Hold it, hold it, Samet says that he can't find an effect, even itsy bitsy effects from ozone precursor and carbon monoxide, something the other EPA favorite researchers say are killing thousands? Samet is not helping the EPA here. What about those dastardly pollutants? We scientists and particularly toxicologists are smiling to see Samet make a fool of himself and by adoption of this weak and deceptive epidemiology, the EPA doesn't look too good either. This is the kind of research the EPA has been using in air pollution regulatory policy now for years.**

--"We did not find an effect of ozone levels on the overall rate of death from all causes or from cardiovascular and respiratory causes during the full year periods. Ozone levels were positively associated with mortality rates during the summer months when ozone levels were highest, although the 95 percent posterior interval extended into the range indicating no effect of ozone levels on mortality." **Might this non-Johns Hopkins man who owns no jacket with arm patches translate for the benighted—Samet says even ozone doesn't have a death effect in his study. Score so far on this paper—rational skeptics for people in search of truth 3, EPA and Samet 0.**

--"We found no evidence that key socioeconomic factors such as low socioeconomic status affect

the association between PM10 and the risk of death in linear regression models." **Some might be surprised to know that Samet works at a School of Public Health and all Public Health research for the last 20 years has shown clearly that there is a socioeconomic effect that produces premature deaths. Skeptics now 4 and running away, EPA and Samet still 0. Socioeconomic noise cancels out air pollution effects; that's the way the epidemiologists put it.**

--"Our analysis also did not address the extent to which life is shortened in association with daily exposure to the various pollutants." **Well golly Dr. Samet, everyone dies, how can you talk about death effects if you don't measure whether deaths are premature? Skeptics 5, Samet and EPA still 0.**

Additional comments by this author:

1. The rate of death changes in Samet's studies are less than 1%, which is epidemiologically meaningless and shows no respect for the relative risk of 2 (100%) or more, that all cohort studies have to show in order to be able to assert effect. Little effects, even in studies with good confidence intervals and lots of power, are still empty studies, make work exercises. Samet's study was a nothing, yet it got published in the *New England Journal,* so one must wonder about political and environmentalist agendas up in Boston. I suppose they are neutral on the environment and always demand valid research in support of political agendas. I suppose.
2. The study fails to age/sex adjust for the important analysis—premature death. How did Samet get published? Samet is asserting proof of effect at less than one two hundredth of what is required in epidemiology. Then he says he didn't bother with measuring whether air pollution caused premature deaths. This research is about acute death affects? At non-toxic pollution levels? There is no plausible biologic science to support the idea that non-toxic air pollution kills people. Samet is beyond redemption. He's in scientific denial, or he works for the EPA agenda and he will be funded until he is old and gray.
3. Low relative risks, below 1.2, are the results in Samet's studies and all the other EPA health effects studies. One study goes above 1.2, the Dockery 1993 smaller study at 1.26, since recalculated by Enstrom in his article, Enstrom 2005 to 1.13. Such weak and minimal findings are unacceptable for publication, much less serious EPA policy making. The EPA and the studies misuse the term statistical significance, trends or association if they mean proof. There is no proof in any of these studies of an air pollution health effect. These studies prove nothing in the relative risk ranges of less than 1.3, particularly in cohort studies of death certificates that are subject to serious confounding.
4. The failure by Samet to find any effect, even these minimal effects, from other air pollutants like nitrous and sulfur oxides (ozone precursors), ozone, and carbon monoxide should give the EPA cause to wonder about any further attempts to impose new ambient air standards. The EPA has noticeably ignored Samet conclusions about these pollutants, why?
5. Samet's assertion that socio economics do not effect death rates is a an extraordinarily faulty conclusion for a public health researcher, since his study only looked at average area incomes for the twenty cities; and there is a vast body of

public health research that shows that socioeconomics independently are a significant factor in life expectancy. (Wong 2002, Fitzpatrick 2001, Lantz 1998).

6. Socioeconomics is a factor and would nullify the signal from air pollution effect, and could even be a cofactor in another way by causing poor indoor air quality from substandard housing and a higher rate of smoking along with a higher rate of underreported smoking. For example the poor have outdoor jobs where they can smoke more, and culturally they may be much heavier smokers with more inhaling, a potential confounder. Such confounding might explain the Ohio and West Virginia data from Pope 1995. That's why relative risk has to be set high, to avoid the effect of confounders not seen or understood.

The Samet article includes cautionary notes on the limitations of the study's methodology. His caveats are applicable to the all the previously mentioned Pope and Dockery, favorite EPA studies on air pollution health effects:

1. "For the pollutants measured on an hourly basis we calculated the 24-hour average." **Toxicologists cringe at that one.**

2. "If the pollutants were measured at multiple locations in a metropolitan area, we averaged the data." **Remember the basic principles of toxicology, if you're downwind from an air pollutant you're safe, how can he say these things with a straight face. You have to know the patient and the toxin and the dose to know anything much about the science. Population studies are very crude at non-toxic levels of exposure.**

3. "Since the Environmental Protection Agency requires levels of PM 10 to be measured only every six days, data for ozone and other pollutants were generally more available on more days." **Good grief, this is a sham, a toxicology study with exposures every so often in sub toxic ranges.**

4. "We analyzed the effect of the day on which the pollution data were obtained (the current day, the day before, or two days before) on the association with mortality rates. The overall effect did not vary with the lag interval selected. We report data for a one day lag between pollution variables and mortality." **This is the place where Dr. Samet shows he doesn't know anything about death. You could be sick to death in a hospital and I can keep you alive indefinitely until the family gives up—where do those cases fit in Dr. Samet's arbitrary lag time of one day? What about people who die in a bed at a nursing home and haven't been outside in two years? These public health wonks and economists who hate dirty air do research as if a death certificate signed by the local GP is a piece of reliable data on the health effects of air pollution. They are in dreamland.**

Then Samet says they found a temporal-causal relationship -- astounding! He didn't find a causal relationship, but he can find a temporal relationship. Did he dredge and dredge until he found something to point at? What's he talking about? Who's to know when the blips in the data are differences of less than 1%? That's not about cause of death, that's about political agendas and a polemic dressed up as science that causes public anxiety.

The good Doctor continues.

5. "Data on levels of PM 2.5 (small particulates) are not yet available nationally, since a monitoring network for particles in this size range is currently being implemented." **This writer believes that Dr. Samet is working the agenda for the "annuity." Small particulates are an annuity for the EPA and air pollution researchers because, along with ozone, dust will never go away. Those air pollution demons assure EPA power into the distant future and more regs and anxiety. Dust is bad. Dust is always going to be there. It's the perfect air pollutant for the EPA.**

Samet and others in the air pollution junk science club just use the PM 10-micron data that is measured every six days as a surrogate for PM 2.5. The supportive press and academic colleagues forgive such a lapse since they are working on the agreed upon agenda.

6. "Our analyses also did not address the extent to which life is shortened in association with daily exposure to the various pollutants."

Extraordinary. If the endpoint is a death effect, then the study must analyze premature death in mortal man and assess acute events as a measure of effect and endpoint for acute and/or chronic disease. To determine premature death effect, age and sex adjusted death rates are the accepted methodology, but Samet is just doing death rates and he gets published in the *New England Journal of Medicine*? Politics and the right agenda trump science and peer review?

7. "The finding that the association between PM 10 levels and the risk of death was strongest for cardiovascular and respiratory causes of death is consistent with the hypothesis that persons made frail by advanced heart and lung disease are more susceptible to the adverse effects of air pollution."

Again they didn't show that at all, they showed less than a 1% effect on death rates. I thought these people were dying of air pollution caused illness, not acute effects of air pollution, which at current levels couldn't kill a canary. What gives? What gives is that Dr. Samet is clueless because he's a numbers cruncher for the EPA in cahoots with his friends in the spic and span air society. I know why people die and it isn't from air in America, or even from Air America. Air pollution comes in many forms but we are obligated to live with toxicology science, not anxiety. Living organisms don't die for the thought of a smoggy day or from a bad smell. Dr. Samet and his cottage clack of air pollution hand wringers should go to a hospital and see how and why people die before they do these desk analyses of death certificates.

Despite these caveats the Samet research group asserts in the conclusion of their paper: "Our analyses provide evidence that particulate air pollution continues to have an adverse effect on the public's health and strengthen the rationale for limiting levels of respirable particles in outdoor air." **Samet says nothing about the significance of their research showing no death effect from ozone, carbon monoxide, sulfur and nitrous oxides. That would certainly disrupt current EPA policy, and he avoids an admission that the relative risks and death rate changes he found do not reach epidemiologic significance.**

This study by Samet is sham epidemiology/science, junk science with lipstick, and the deception and "newspeak" harkens back to junk science in the service of the King or the current tyrant. Pope, Dockery and Samet are the officials/magicians/astrologers/conjurers in the EPA court, providing the EPA regent with needed "expertise" to justify the latest edict.

Briefly we will discuss below Dr. Samet's mentors, the EPA's favorite air pollution haters, Drs Dockery and Pope, who work together and change places on the authors lists of their papers.

The Six City and Pope Studies?

Dockery (1993) and Pope (1995) did studies that were the model for the Samet study discussed above. The studies did do better than Samet, in that they measured relative risk of premature death by studying death rate with age sex adjusting. Both Dockery and Pope were unable to show significant relative risk of health effect. The Pope and Dockery studies were used in the mid 1990s to justify EPA Director Browner's "emergency" new ambient air quality standards on ozone and other pollutants. The resulting cost was estimated by the Center for Study of American Business at Washington University, St. Louis, at more than 100 billion. The Browner action was taken unilaterally, in spite of protests from many agencies within the government and without the approval or support of EPA internal experts. This action was taken without proof of a health effect, since Pope and Dockery never showed an acceptable relative risk. They were limited again to Samet's "associations" and trends within meaningless ranges below a relative risk of 1.3.

There is a greater relative risk of whole milk causing lung cancer than the relative risk that the EPA has shown for air pollution. One might say that's because of some confounder—well duuuh, that's why relative risk has to be above a threshold of 2 and some say 3, so confounders don't make the epidemiologist look confounded. Samet, Pope, Dockery don't care, they're on a roll and have the support of the environmentalist zealots, and the EPA (whoops, that's redundant). Call public relations, the research shows air pollution is killing thousands. It causes CANCER.

This paper points out that the EPA and the researchers are cheatin', and Dr. K. Popper, famous philosopher of science favorably cited by the Supreme Court in the *Daubert* decision, says that science must be more serious and reliable than politics. Popper asserts that science must be based on proofs that are reliable. Popper even talks about what the air pollution research by Pope, Dockery and Samet and the spic and span society is—Popper says some "science" is so bad it can't be falsified. How does one falsify something that means nothing? Associations at the edge of or in the midst of nothingness is what Pope's and the other health effects studies assert should be the basis for society wide regulatory regimes. Breathtaking—no pun

intended.

The EPA says that air pollution kills thousands, because air pollution kills thousands. That is a tautalogy, a common tool for junk scientists. IT IS BECAUSE IT IS. I write here to tell the EPA that their anxious pursuit of clean air is more about politics and power and anger with modern industrial society that is already cleaning up the air, more about the religion of environmentalism. That's why the crisis, without the deaths or the science is a political or a polemic tool, not science. Not nice to fool with science that way, particularly when there is a Federal mandate that the EPA insist on scientific integrity for policy making. The EPA should not be in the business of ginning up false crises and scaring mothers that their kids are going to suffer from the air just so that the bureaucracy will thrive at the Federal and State level.

The EPA cannot claim to be unaware of the failure to prove health effects by the insignificant level of relative risk in the Pope and Dockery studies. These are the most basic of epidemiologic rules. And no subsequent studies have rehabilitated the failures of the Pope and Dockery studies. Samet, as described above, just repeated the same mistakes and came to up with the same lack of proof of health effect, unjustified conclusions and excessive and activist recommendations.

The barriers to a good study on health effects of air pollution for Dockery and Pope were the same as for Samet,

1. mobile populations,
2. unreliable, non-continuous and fixed monitor information,
3. no monitor information on some pollutants all the time (2.5 micron particles for example) or part of the time (10 micron and others),
4. an attempt to assess long term chronic health effects of air pollution by death studies, an acute phenomenon,
5. death certificates and raw death data used without autopsies,
6. inside air quality ignored for populations living indoors, particularly during old age, advanced medical illness, and terminal illness,
7. But most of all, no biological plausibility because the deaths are in the setting of non-toxic levels of air pollution (the inane straight line effect toxicology of the EPA cannot continue to get a pass—it is advocacy at the expense of science).

The EPA in assessing the air pollution effects studies must revive Bradford-Hill Criteria for toxicology

The Bradford Hill (BH) criteria for toxicology are elementary, and establish biological plausibility for toxin effects. They require the toxicologist to establish plausibility, dose effect, reproducibility, time relationship, and a pattern of predictable and observable effects. Sounds like good science, but that's only part of it. Karl Popper was referenced above as the guru of the philosophy of science, and master or curator of scientific principles. The Popper legacy of science rules are referred to reverently in the Supreme Court opinion in the Daubert v. Merrill Dow Case [509 U.S. 579 (1993)] on admissibility of scientific testimony. Falsifiability is the key. To be true science one must submit to the test of being proven wrong. Pope and Dockery study

results can't be falsified because they don't even allow a legitimate assertion of proof. They are tools in the game of politics, not in the game of toxicology. The EPA is required by common sense and federal statute to apply the BH criteria in air pollution studies, and all other toxicology work, but instead this wildly deceptive use of small changes within insignificant ranges of effect is souped-up to become the reason the EPA must act, now, immediately, to save lives. The EPA is saving itself, but the air pollution regulations are not saving any lives because the research would show the lives lost with valid epidemiology, and it doesn't.

The only reason that the EPA can create a crisis from the Pope or Dockery studies if it holds its nose and just projects to the whole population of the United States, then relative risk of less than 5% becomes thousands of deaths, even though it fails to show proof of one death caused by the toxicity of air pollution. Not one death.

If the biological plausibility of air pollution causing disease and death consistent with the BH criteria was established or could be established, then EPA and air pollution health effects researchers like Pope, Dockery and Samet could rest with their laurels. If air really were a killer or a toxin, we wouldn't see these weak cohort studies from the EPA with itsy-bitsy relative risks, and the argument would be over.

The EPA is not the national agency or institute for the arts, culture, pleasantness and good smells, it has a serious public health responsibility and a federal mandate to find toxins with legitimate science, promulgate appropriate solutions for the public benefit and then assess the effectiveness of what it has done. None of those steps are being taken in the air pollution policy making of the EPA.

The air pollution health effects studies in America will never be able to show the required relative risk of 2 or 3. What was the EPA role in such deception?

The idea that seems to control the EPA policy making on air pollution in the past 15 years is--ignore methodology and statistical problems, science be damned, move on to the grand program of air purification. Find the ultimate terrible pollutant that will never go away, even with all our regulations. That is why small particulates are so promising for the EPA, enough so that these health effects studies talk about small particulates without measuring them, or measuring them in only one part of the study and not everywhere. The project of demonizing small particulates is reflected in the Samet study. He makes strong assertions with extraordinarily weak evidence, but he goes to the meetings, he knows what the EPA is concerned about. With EPA leading and frequently funding the crusade—science and truth casualties are acceptable. Small particulates are the worst crisis in the history of air pollution, they might cause CANCER.

I grew up and still live on a farm. I consider dust a reality that cannot be regulated away, just like ozone is part of the Smoky Mountains. There is a form of air pollution that is now being generated by the EPA in its ozone and small particulates crisis project—it is composed of dust, water, methane, and biological particulates.

Joseph Shumpeter said that the first casualty of a commitment to an ideal is the truth. The second casualty, this author asserts, is the unwary taxpayer and public that depends on responsible government. Solzhenitsyn said "The simple step of a courageous individual is not to

take part in the lie. One word of truth outweighs the world." The EPA has become a slave to the lie of junk science in health effects research because the agency is devoted to its own importance and the importance of its religious and political agendas. EPA dredges up and makes icons of the precautionary principle, the small numbers/large projections lie, small trends within meaningless relative risks in populations studies, the refusal to recognize basic toxicology concepts. The EPA is a rogue agency in need of a stand down and close internal inspection.

The Killer Smog

In *The New England Journal of Medicine, Dr.* C. Arden Pope, clean air activist, and one of the EPA's all time favorite air pollution health effects researchers, describes killer air in Belgium in 1930, Pennsylvania in 1948, and London in 1952 -- and uses those incidents as examples of why he thinks there is good reason to pay attention to a study in that issue of the *Journal* that claims to show a causal relationship between non-toxic air pollution and children's pulmonary functions. Again the study he is supportive of shows no epidemiological proof, just "associations," which are nothing more than statistical cluster puffs in population studies subject, as pointed out above, to bias and confounders. But the key is the study includes two important things for environmentalist zealots, children, and air pollution. Most importantly this study, like all the air pollution health effect studies, is working in insignificant causation ranges of effects so Pope and the EPA can talk about little bitsy trends and associations and urge that something be done before children die on playgrounds. They talk of these numbers exercises like they foretell an apocalypse. *Gather the elderly and children and go seek shelter from the air, says Dr. Pope, an economist who got in the air pollution health effects business because he hated the air in Utah—imagine if he had lived in New Jersey. Dr. Pope advises---Stop breathing, if you must.*

People do not go out into the streets of America, choke and die. The days of the people of London and Pittsburgh wearing dark clothes to mask the effect of soot and smoke are gone. The public health hanky battalion wants Americans to think air is killing their children and old folks, but in America ambient air pollution did not kill anyone, last week, last year, or in the last ten years. The panicky talk has to stop and the EPA must stop being the sponsor of the lie. The medical journals have to put their scientist hats back on and stop wringing their hands about nonsense environmental crises. The EPA is so busy these days frightening people about their rat studies and the imagined effects of so many things. Hardly enough time in the day to pursue air pollution, except the EPA has lots of staff and lots of money and much energy and religious devotion to the cause.

EPA Policy and Regulation Activity

Fredrick Bastiat is known for his "law of unintended consequences," best exemplified as the analysis of the Paris shopkeeper's broken window. Bastiat made a common sense observation that when government or individuals choose to spend money or act, it produces desired and undesired effects, always making a ripple within the society and economy.

Let us propose to the EPA that if asthma deaths are predominately in young adult black males in America because of poor compliance (McFadden 1997), due to cost and availability of asthma treatment for disadvantaged adult black males or some other socio-economic or political problem, the EPA would be foolish to work on parsing senseless air quality regulations in

preference to better asthma health care. The EPA would not be a party to such nonsense, would it, to relieve the anxiety of anxious environmentalists or satisfy the EPA staff's need for power and control?

There are no free regulatory actions. Every choice has multiple consequences, and government interventions have effects unforeseen. The EPA takes taxpayer dollars for every jot and tittle, every phone call, every new grand idea of every zealous bureaucrat. Every dollar spent for the EPA's ideal of pure air comes from somewhere and is taken from somewhere else.

The EPA is charged with responsible health effects research and policy making. The questions raised in the mid 1990s and now are the same:

1. If relative risk is a well-known measure of cause and effect in epidemiology, why does the EPA allow relative risk below acceptable levels of proof to influence policy making?
2. Considering that EPA regulatory activity is tremendous burden to the economy, and the air regulations have a cost effect measured in billions per year taken from the taxpayer. If socioeconomic factors are an undeniable influence on quality of life and life expectancy, then can weak and unacceptable health effects epidemiology as described above, be excused for some abstract ideal of pure air?
3. Can studies that measure acute events in any way be considered studies of cumulative health effects? Are these death studies that Pope and the other air pollution researchers insist on basically flawed and deceptive. The answer is yes.
4. If some of the studies can't eliminate confounders, does the EPA have the authority to impose an onerous regulatory regime on the American society on the theory that cleaner air is a worthwhile, even if it doesn't have any effect on health?

Enstrom Particulate Air pollution Health Effects Study of 50,000 elderly Californians.

Dr. James Enstrom, in the attached article found in appendix A, studied deaths in elderly Californians in 25 counties. He found that the relationship between fine particulates and mortality was very weak during the 1973-2002, particularly after 1982. He also reviewed the cohort studies on health effects of fine particulates and mortality by Pope, Dockery, McDonnell, and Lipfert, and found that their results were fairly similar to his, with the weakest health effects being present during the most recent years.

Enstrom finds:

1. The relative risks, age and sex adjusted and homogenized, are close to 1.00 in his and the other death studies (Pope, Dockery, McDonnell, Lipfert) he reviews in Table 10—there is no proof of health effect shown from particulate air pollution in his or the other studies.
2. Pope's year 2000 16 year follow up to the earlier (Pope 1995) study of the same cohort (Pope 2002) shows a declining cumulative risk from 1.07 to 1.04, first half to

second That means to all but the innumerate that the relative risk in the second decade is well below 1.04. **Hello Dr. Pope, Helloooo EPA.**

3. Enstrom points out there are substantial geographic variation between the California populations of his study and Pope's Ohio, Kentucky and West Virginia data. The potential for confounders should be considered. **I know something about that, and people in those states aren't the same as people in Enstrom's study. They might live different lives from their fellow citizens in Lala land. That's what homogenizing and sampling in epidemiology is all about. Without the data from those three states, Pope's studies would be more epidemiologically insignificant than they are, if that were possible. So much for avoiding cherry picking and the admonition to chip off the edges of the data to norm a cohort analysis.**

The important points of the Enstrom study:

.

1. Deaths and air pollution relative risks were assessed for 25 California counties, a cohort of 50,000 elderly Californians, and 39,000 dead before the end of the study in 2002. The relative risks were measured with proper confidence and homogeneity.
2. Relative risk found was extremely small and insignificant, 1.04 in the first part of the study (1973-1982), then relative risk of death from air pollution disappeared altogether in the second part of the study (1983-2002). **Which will it be EPA, a crisis or salvation from killer air.**
3. For the entire period the relative risk was 1.01 **Pulleeez, 1-% risk? That's a relative risk of 1.01. I am closer than that to being rich and good looking, like Michael Jordan. The results would have to be 2.00 to be proof of any health effect, 1.00 is no effect**.)
4. **This Enstrom study, like all the other studies that the EPA uses to analyze health effects, and supposedly to study small particulate effects, is limited by the lack of PM 2.5 micron monitors before 1979 and only limited monitors after.**
5. No increased death effects of any kind were shown in the counties with higher levels of air pollution, eliminating any dose response effect (a favorite rhetorical tool of the EPA researcher group), that, some of the higher pollution counties had lower relative risks. **So is air pollution good for you if you live in California? In this range of relative risk absence of trend is meaningless but Dr. Enstrom does the prescribed exercise, since the air pollution cabal likes to do trending and associations. The idea of a trend within an insignificance is interesting to consider, for fun, but not for science.)**
6. Table 10 in Enstrom's paper shows a comprehensive review of comparable relative risks from large (Pope, Enstrom) and small (Dockery, McDonnell, Lipfert) studies, showing that only the Dockery study published in 1993 in a small cohort shows a relative risk above 1.1 at 1.15. All the other studies show relative risk similar to Enstrom, in the range of 1.07 or less.
7. In table 10 a number of the confidence intervals cross 1.0, the cumulative relative risk of the Pope study for the second half is lost in the failure to separate out the second half, indicating there is a reason to believe that in the second half of his study 1990-98, Pope had a relative risk approaching an insignificant 1.01. **I worry, sort of, about Pope hiding this bad trend downward of an already weak relative risk. Could one suppose he has revealed this problem to his friends at the EPA?**

Suresh Moolgavkar

It would not be practical here to cover all the writings of Suresh Moolgavkar on the epidemiologic and methodology problems he identifies in the EPA air pollution health effects research and policy making, and this writer does not understand some of the subtleties. Dr. M's brain and pen are too capable for an adequate treatment here, by a mere emergency physician. Dr. Moolgavkar's recent in depth review and critique of EPA particulate and air pollution research and policy making is in Appendix B.

Moolgavkar 2005 wrote a commentary on Enstrom's paper for *Inhalation Toxicology* discussed above (see second part of App. A). He asked the rhetorical question "Can contemporary epidemiological and statistical tools reliably detect miniscule risks, particularly with strong risk factors as potential confounders?" (**Dr. Moolgavkar is too kind. He politely avoids exposing the junk science, the obvious, that miniscule risks in a cohort study like the results in the Pope, Dockery and other studies show no health effects at all and talking about trends in those ranges is silly.)**

Moolgavkar objects to the methodology of proportional hazards modeling because "it is highly unlikely that proportionality of hazards would hold over the entire period of time covered by these studies." (The long term air pollution health affects studies). He asserts that it can be argued that "the SO 2 effect wipes out the PM signal in joint pollutant models." He does not even address the Samet study showing no SO 2 effect, so even that problem may ignore the more basic one that is so apparent—there is no detectable causal effect between air pollution and death. Dr. M is operating with the assumption that SO2 still is on the top of the list of bad pollutants. No doubt it is more toxic than others, but again, we must repeat the toxicology commandment—**the dose makes the toxin.** The air pollution health effect studies relied on by the EPA are ridiculously weak and are used as silly substitutes for a lack of laboratory proof that the current air conditions cause disease. The health effects research of Pope Dockery and Samet is just an exercise in the traditional deception of the "data dredge," the tool of crisismongers.

What is the point of quibbling about miniscule, below threshold of proof, differences in a cohort death study, some slavish devotion to arithmetic? I benefit, I suppose from not liking higher math, in this circumstances, that's why I focus on the medicine and the proper analysis of death studies and why people die.

Moolgavkar (2005 See App. B) wrote a lengthy review and criticism of EPA policy in *Regulatory Toxicology and Pharmacology* that exposes the epidemiologic and toxicological problems of the EPA air pollution health effects research discussed above.

Moolgavkar asserts: "evidence fell far short of supporting a causal association between particle mass concentration and human health." He goes on "the results of observational epidemiology studies can be seriously biased, particularly when estimated risks are small, as is the case with studies of air pollution. The Agency (EPA) has largely ignored these issues." "I conclude that a particle mass standard is not defensible on the basis of a causal association between ambient particle mass and adverse effects on human health."

Although Moolgavkar allows that the EPA may be bending the science in an attempt to pursue the precautionary principle on particulates, the precautionary principle under a mandate of good science in the public interest, is not good policy. It is the default position for making concerns,

feelings and aesthetics into the basis for regulatory actions that cost society billions for compliance. However no sandal-footed environmentalist gang of enviro-religious concerned citizens can allow the EPA to reject science.

The EPA is prohibited by federal mandate from ignoring science in the pursuit of the precautionary principle. The precautionary principle is anti-science and irrational by definition. Health effects not shown scientifically trumps feeling, concern and governmental overreach. The EPA is mandated by federal law to halt the overreach of the air pollution crisis crusade until it can resuscitate science in the public interest.

Author--John Dale Dunn MD JD

Endnotes

Samet 2000 Samet JM, Dominici F, Curriero FC, et.al. Fine particulate air pollution and mortality in 20 U.S. cities, 1987-1994. *NEJM 2000; 343:1742-9.*

Wong 2002 Wong JD, Shapiro MF, Boscardin WJ, et. al. Contribution of major diseases to disparities in mortality. *N Engl J Med* 2002;347:1585-92.

Fitzpatrick 2001 Fitzpatrick R. Ed. Social status and mortality. *Ann Intern Med* 2001 134;10:1001-2.

Lantz 1998 Lantz PM, Lepkowski JM et. al. Low income was an independent risk factor for premature death after controlling for health behaviors. *JAMA* 1998; 279:1703-8.

Dockery 1993 Dockery DW, Pope CA 3d, Xu X, et. al. An association between air pollution and mortality in six U.S. cities. *N Engl J Med* 1993;329:1753-9.

Pope 1995 Pope CA, Thun MJ, Manboodiri MM, et. al. Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. *Am J Respir Crit Care Med 1995;151:669-74.*

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McDonnell 2000 McDonnell WF, Nishino-Ishikawa N, Petersen FF, et.al. Relationship of mortality with the fine and coarse fractions of long-term ambient PM10 concentrations in non-smokers. *J Exper Environ Epidemiol* 2000;10:427-436.

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Pope 2004 Pope CA. Ed. Air pollution and health -- good news and bad. *N Engl J Med* 2004 351;1132-1134.

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Enstrom 2005 Enstrom J. Fine particulate air pollution and total mortality among elderly Californians, 1973-2002. *Inhalation Toxicology* 2005; 17:803-16.

Moolgavkar 2005 Moolgavkar S. Let. Fine particles and mortality. *Inhalation Toxicology 2006;18:93-4.*

Moolgavkar 2005 Moolgavkar S. A review and critique of the EPA's rationale for a fine particle standard. *Reg Tox Pharm* 2005; 42:123-44.

Appendix B

Abstract only with one table added.

Inhalation Toxicology, 17:803–816, 2005

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DOI: 10.1080/08958370500240413

Fine Particulate Air Pollution and Total Mortality Among Elderly Californians, 1973–2002

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Fine particulate air pollution has been associated with increases in long-term mortality in selected cohort studies, and this association has been influential in the establishment of air quality regulations for fine particles ($PM_{2.5}$).However, this epidemiologic evidence has been questioned because of methodological issues, conflicting findings, and lack of an accepted causal mechanism. To further evaluate this association, the long-term relation between fine particulate air pollution and total mortality was examined in a cohort of 49,975 elderly Californians, with a mean age of 65 yr as of 1973. These subjects, who resided in 25 California counties, were enrolled in 1959, recontacted in 1972, and followed from 1973 through 2002; 39,846 deaths were identified. Proportional hazards regression models were used to determine their relative risk of death (RR) and 95% confidence interval (CI) during 1973–2002 by county of residence. The models adjusted for age, sex, cigarette smoking, race, education, marital status, body mass index, occupational exposure, exercise, and a dietary factor. For the 35,789 subjects residing in 11 of these counties, county-wide exposure to fine particles was estimated from outdoor ambient concentrations measured during 1979–1983 and RRs were calculated as a function of these $PM_{2.5}$ levels (mean of 23.4 $\mu g/m^3$). For the initial period, 1973–1982, a small positive risk was found: RR was 1.04 (1.01–1.07) for a 10-$\mu g/m^3$ increase in $PM_{2.5}$.For the subsequent period, 1983–2002, this risk was no longer present: RR was 1.00 (0.98–1.02). For the entire follow-up period, RR was 1.01 (0.99–1.03). The RRs varied somewhat among major subgroups defined by sex, age, education level, smoking status, and health status. None of the subgroups that had significantly elevated RRs during 1973–1982 had significantly elevated RRs during 1983–2002. The RRs showed no substantial variation by county of residence during any of the three follow-up periods. Subjects in the two counties with the highest $PM_{2.5}$ levels (mean of 36.1 $\mu g/m^3$) had no greater risk of death than those in the two counties with the lowest $PM_{2.5}$ levels (mean of 13.1 $\mu g/m^3$). These epidemiologic results do not support a current relationship between fine particulate pollution and total mortality in elderly Californians, but they do not rule out a small effect, particularly before 1983.

Table ten is present as originally published in a pdf file of the article. Attached.

TABLE 10 Relative risk (RR) and 95% confidence interval (CI) for long- term all- cause mortality per 10- μ g/ m^3 increase in $PM_{2.5}$ for U. S. cohort studies based on $PM_{2.5}$ data, circa 1980

$PM_{2.5}$ Study characteristics

Study (author, year)
Data period/ Mean (range)/ (μ g/ m 3)/ Cohort geographic definition/ Follow- up period/
Mean entry age for period/ Number entered in cohort/ Deaths in follow-up period/ RR (95% CI)
Males
Dockery et al., 1993 1979– 1985 19 (11– 30) 6 U. S. cities 1975– 1989 _ 50 3671 [a] 830 [a] 1.15 (1.02– 1.30) [b]
Pope et al., 1995 1979– 1981 18 (9– 34) 50 U. S. SMSAs 1982– 1989 57 130,310 [a] _ 12,400 [a] 1.07 (1.03– 1.11) [b]
McDonnell et al., 2000 1973– 1977 32 (17– 45) 9 CA airsheds 1976– 1992 58 _ 1347 _ 375 1.09 (0.98– 1.21) [b]
Lipfert et al., 2000 1979– 1981 24 (6– 42) 42 U. S. counties 1975– 1981 51 26,067 _ 4600 [c] 0.95 (0.89– 1.01) [c]
1982– 1984 22 (8– 41) 1982– 1988 57 _ 21,467 _ 6100 [c] 0.94 (0.90– 0.98) [c]
1982– 1984 22 (8– 41) 1989– 1996 63 _ 15,367 _ 5765 [c] 0.89 (0.85– 0.95) [c]
Pope et al., 2002 1979– 1983 21 (10– 30) 61 U. S. SMSAs 1982– 1998 57 _ 159,000 [a] _ 36,000 [a] 1.05 (1.01– 1.10)
Enstrom, 2005 1979– 1983 24 (11– 42) 11 CA counties 1973– 1982 66 15,573 4701 1.03 (0.99– 1.07)
1979– 1983 24 (11– 42) 1983– 2002 74 10,872 8831 0.97 (0.95– 1.00)
Females
Dockery et al., 1993 1979– 1985 19 (11– 30) 6 U. S. cities 1975– 1989 _ 50 4440 [a] 599 [a] 1.12 (0.96– 1.30) [b]
Pope et al., 1995 1979– 1981 18 (9– 34) 50 U. S. SMSAs 1982– 1989 57 164,913 [a] _ 8365 [a] 1.06 (1.01– 1.12) [b]
McDonnell et al., 2000 1973– 1977 32 (17– 45) 9 CA airsheds 1976– 1992 58 _ 2422 _ 568 _ 1.00 (assumed)
Pope et al., 2002 1979– 1983 21 (10– 30) 61 U. S. SMSAs 1982– 1998 57 _ 200,000 [a] _ 24,000 [a] 1.02 (0.98– 1.06)
Enstrom, 2005 1979– 1983 24 (11– 42) 11 CA counties 1973– 1982 65 20,210 4094 1.05 (1.01– 1.10)
1979– 1983 24 (11– 42) 1983– 2002 73 16,116 10,815 1.02 (0.99– 1.04)
Both Sexes
Dockery et al., 1993 1979– 1985 19 (11– 30) 6 U. S. cities 1975– 1989 _ 50 8111 1430 1.13 (1.04– 1.23) [b]
Pope et al., 1995 1979– 1981 18 (9– 34) 50 U. S. SMSAs 1982– 1989 57 295,223 20,765 1.07 (1.04– 1.10) [b]
Pope et al., 2002 1979– 1983 21 (10– 30) 61 U. S. SMSAs 1982– 1998 57 _ 359,000 _ 60,000 1.04 (1.01– 1.08)
Enstrom, 2005 1979– 1983 24 (11– 42) 11 CA counties 1973– 1982 65 35,783 8795 1.04 (1.01– 1.07)
1979– 1983 24 (11– 42) 1983– 2002 73 26,988 19,646 1.00 (0.98– 1.02)
[a] Obtained from supplementary data (Krewski et al., 2000). [b] Recalculated from published data (US EPA, 2004). [c] Obtained from the author.
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Fine Particles and Mortality

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In an interesting paper in a recent issue (vol 17, issue 14) of the journal, Enstrom examined the association between fine particulate matter (PM) pollution and mortality in a cohort of elderly Californians. The analyses used proportional hazards regression and after adjusting for age, sex, cigarette smoking, and other potential confounders, Enstrom concluded, "These epidemiologic results do not support a current relationship between fine particulate pollution and total mortality in elderly Californians, but they do not rule out a small effect, particularly before 1983." Enstrom's analyses were based on a sub-cohort of individuals enrolled in the first Cancer Prevention Study (CPS I) conducted by the American Cancer Society (ACS). Enstrom's conclusion is consistent with the conclusions of a cohort study among veterans conducted by Lipfert et al. (2000), but is at odds with the results from analyses of the second ACS cohort (CPS II) by Pope and others (Pope et al., 1995, 2002; Krewski et al., 2000), which reported statistically significant associations between fine particulate pollution and mortality.

Every epidemiological study has weaknesses and limitations and, undoubtedly, both proponents and skeptics of the 'fine par-ticles cause death' thesis will find much to criticize in the studies that do not support their conclusions. These discrepant results raise an important question, however. Can contemporary epidemiological and statistical tools reliably detect miniscule risks, particularly with strong risk factors as potential confounders?

All the cohort studies referred to above use proportional haz-ards modeling for data analyses. But is proportional hazards really the appropriate tool for these analyses? First, it is highly unlikely that proportionality of hazards would hold over the en-tire period of time covered by these studies. Statistical tests for departures from proportionality of hazards have low power. En-strom states that, in his analyses, these tests failed to reject pro-portionality of hazards. However, his finding of a higher relative risk associated with fine particles over the period 1973–1982 is inconsistent with proportionality of hazards over the entire

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period of the study. Even if proportionality of hazards were to hold for exposure to fine particles, we know that it most defi-nitely does not hold for cigarette smoking, a strong risk factor and a potential confounder of the PM mortality association. For example, we know that, for a given daily level of smoking, the relative risk of lung cancer is strongly dependent on duration of smoking. Moreover, when smokers quit, the relative risk for

mortality declines over a period of many years, and not virtually instantly as is assumed by proportional hazards. What influence does this manifestly incorrect model for a strong confounder have on the estimates of air pollution effects? Similarly the use of a proportional hazards model to adjust for age of entry into studies is also suspect.

I have discussed the original CPS II study (Pope et al., 1995) and reanalyses (Krewski et al., 2000; Pope et al., 2002) in de-tail elsewhere (Moolgavkar, 2005). I note here, however, that the reanalysis by Krewski et al. (2000) of the original (Pope et al., 1995) study (which considered no pollutant other than PM), showed quite clearly that the pollutant most strongly as-sociated with mortality was not PM but SO_2.In fact, when SO_2 was considered along with PM in the model for all-cause mor-tality, the coefficient for sulfates was reduced to less than a third of its original value, that for fine particles was reduced to a sixth of its original value, and both became statistically insignif-icant. It is also of interest to note that consideration of spatial correlations attenuated the PM coefficients to a much greater extent than the coefficients for SO_2.Given the much stronger and more robust association of SO_2 with mortality in the CPS II reanalyses, I find it surprising that this study continues to be taken as providing strong support for the PM mortality asso-ciation. It can be plausibly argued on biological grounds that SO_2 could not be causally associated with mortality. But that still does not explain why SO_2 wipes out the PM signal in joint pollutant models. This awkward fact has simply been dismissed as being irrelevant. In a more recent study of the CPS II cohort that doubles the follow-up time and triples the number of deaths, Pope et al (2002) reported significant associations between fine particles and oxides of sulfur with all-cause, cardiovascular and lung cancer mortality. Surprisingly, despite the findings in the Krewski analyses that SO_2 was the pollutant most strongly as-sociated with mortality, no joint pollutant analyses were carried

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Appendix C

A review and critique of the EPA's rationale for a fine particle standard

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Received 18 November 2004
Available online 24 March 2005
Abstract
I review the rationale for the Environmental Protection Agency_s 1996 fine particle standard, which was based almost entirely on
the epidemiological data with neither support from Toxicology nor understanding of mechanism. While many epidemiological
papers available in 1996 reported associations between ambient particles and adverse effects on human health, many others did
not and the evidence fell far short of supporting a causal association between particle mass concentration and human health.
The literature appearing after 1996 further complicates the picture. The large studies that have appeared after 1996, such as National
Mortality Morbidity and Air Pollution Study, and the reanalyses of the American Cancer Society II study, report risks that are
substantially smaller than the risks reported in the 1996 Criteria Document and Staff Paper. Moreover, concerns about confounding
by weather, temporal trends and co-pollutants remain unresolved. Other issues having to do with model choice have resurfaced as a
result of reanalyses of critical data to address a glitch in a widely used software package for time-series epidemiology studies of air
pollution. Finally, contemporary examples show that the results of observational epidemiology studies can be seriously biased, par-ticularly
when estimated risks are small, as is the case with studies of air pollution. The Agency has largely ignored these issues. I
conclude that a particle mass standard is not defensible on the basis of a causal association between ambient particle mass and
adverse effects on human health. Such a standard may be justifiable on the basis of the precautionary principle, however. The
Agency could argue that the Science raises concerns about current levels of air pollution, and that reduction of ambient fine par-ticulate
matter mass, if it could be achieved without an increase in the level of the ultrafines, could have positive effects on human
health. If the Agency justifies a particulate matter mass standard on these grounds then the debate over the form and level of the
standard will, for all practical purposes, belong strictly in the Policy arena.

Keywords: Air Pollution; Particulate matter; Criteria document; Staff paper

Appendix D

Here is a previous submission by Dunn on Ozone that is still pertinent.

October 8, 2007

Subject: Comments on OZONE Standards

Submitted via the a-and-r-docket@EPA.gov 10-9-07

Docket ID No. EPA-HQ-OAR-2005-0172

National Ambient Air Quality Standards (NAAQS) for Ozone, 2007.

Comments by John Dale Dunn, MD, JD Policy Advisor, Heartland Institute, Chicago, IL. Member, Board of Scientific and Policy Advisers, American Council on Science and Health, New York, NY.

Corrected and revised final draft submitted 0915 CDT 10-9-07.

1. The EPA ozone science does not justify continued aggressive ozone regulation and a new lower 8-hour standard.

2. The observational air pollution studies and the weak exercise/ozone inhalation studies cited by the EPA show weak associations and relative risk less than 1.5, as well as lab results best described as non adverse. The study evidence cited by the EPA would not be admissible in a Federal Court because it violates basic epidemiology and toxicology scientific rules.

3. The EPA's own Clean Air Scientific Advisory Committee advised in the past that ozone effects research did not show adverse effects and the ozone standard should be left as is.

4. There is no EPA research that shows any benefits from the air quality improvements of the past 20 years. Is it that the EPA doesn't want to report any improvement, for fear it will jeopardize agency funding? Is it evidence that the air pollution wars of the past 20 years were against a PHANTOM MENACE? Are the weak population studies on air pollution weak for a reason--there was no killer air in America?

DISCUSSION

The EPA cited health effects studies are weak on adverse ozone health effects and weak generally on air pollution adverse effects

The Scientific studies discussed in the proposal document are reviewed below. Although the studies are cited by the EPA to justify the ozone standard, they are not what the EPA commentary says they are. They do not excuse the old standard, or justify the new proposed ozone standards because they are a combination of weak observational studies and no-effect intense exercise/high ozone studies.

Commentary on some of the prominent studies:

1 Dockery DW, Pope CA 3d, Xu X, et. al. An association between air pollution and mortality in six U.S. cities. *N Engl J Med* 1993;329:1753-9.

Weak observational study that mentions, but does not control confounders. The results are small effects with relative risks of an insignificant magnitude that is proof of nothing.

2 Pope CA, Thun MJ, Namboodiri MM, et.al. Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. *Am J Respir Crit Care Med 1995;151:669-74*.

Like the Dockery study above, one of the EPA's most important studies for justifying air pollution regulations. This study is another example of weak epidemiology with weak relative risks and no correction for confounders.

Even after the congress passed a law sponsored by Senator Shelby, requiring Pope and Dockery to produce their data sets, they still dodge and feint, and have not complied. Pope and Dockery are still in the inside clique of EPA favored and sponsored epidemiologists. They continue unhindered and well funded by the EPA and other governmental grant sources friendly to an aggressive regulatory agenda.

3 Hrostman DH Ozone concentration and pulmonary response relationships for 6.6 hour exposures with five hours of moderate exercise to 0.9, 0.10, and 0.12 PPM. *American Review of Resp Dis* Nov, 1990; 142: 1158-63.

Even heavy exercise with ozone inspired above current limits shows little ozone effect and no disease. The effect shown was mostly subjective respiratory mechanical effect. Ozone makes air heavy and increases its suspended/solute load.

4 Samet JM, Dominici F, Curriero FC, et.al. Fine particulate air pollution and mortality in 20 U.S. cities, 1987-1994. *NEJM 2000; 343:1742-9.*

Study of cities that claims to know how many days it takes for air pollution to kill someone, then proceeds to find no kill effect from all the air pollution factors, including ozone and ozone precursors, except small particulates, but then admits that the small particle monitor information is not available for the study and that big particles were used as a surrogate. Breathtaking, but published by Dr. Samet's friends

in Boston. Incidentally the EPA on its air web site now has announced that large particles are no longer monitored or controlled because they do not cause adverse effects, but the old studies that concluded the dangers of small particles admit they used large particle monitor data as a surrogate for the small particles, since small particle monitors only became available in the late 1990s.

5 Wong JD, Shapiro MF, Boscardin WJ, et. al. Contribution of major diseases to disparities in mortality. *N Engl J Med* 2002;347:1585-92.

Discussion of confounders in death studies. Apparently has not been read by EPA sponsored and in-house epidemiologists, since the proposal documentation of the EPA makes little mention of the problem of the studies that are relied on—they make assertions without caveats like they were environmental gurus.

6 Fitzpatrick R. Ed. Social status and mortality. *Ann Intern Med* 2001 134;10:1001-2.
Lantz 1998 Lantz PM, Lepkowski JM et. al. Low income was an independent risk factor for premature death after controlling for health behaviors. *JAMA* 1998; 279:1703-8.

None of the studies used by the EPA for air pollution regulatory strategies control well for socio-economic status. Some of the studies do nothing more than mention that average income and education were used over large areas. Very similar to the casual use of wide-area, even regional monitors as measures of exposure to pollution.

7 McFadden ER jr., Warren EL. Observations on asthma mortality. *Ann Intern Med* 1997;127:142-7.

Shows that asthma mortality is in a select group of patients and caused by under-treatment and socioeconomic factors.

8 Mcconnell R, Berhane KT Gilliland F, "Asthma in exercising children exposed to ozone: a cohort study, *Lancet* 359 (2002) 386-91.

Selective reporting of this study ignored the protective effect of ozone, (yes, protective) in the whole cohort while making much of a minimal evidence of detrimental effects in one group--kids who were in three sports. McConnell is part of the Gauderman group that specializes in studying air in Southern California and always finds detrimental effects, even though many times the methodology and the evidence of risk are questionable and weak.

9 Gauderman WJ, Vora H, McConnell R, et al. Effect of exposure to traffic on lung development from 10 to 18 years of age: a cohort study. Lancet (on line) Jan 26, 2007. www.thelancet.com.

This study by the Southern California group had two major problems--1. Very small pulmonary function differences, less than 5%, which is insignificant, and no real negative trend, since the trend line only existed because of one outlier. There was also a high drop out rate. 2. The study measured differences in groups up to 1500 meters, dividing by 500 meters except for a group within 300 meters. Research shows that air quality from roadways is at background by 300 meters. The air quality on Southern California roadways was reported by H. Zhu in *Atmospheric Environment* 2002; 36: 4325-35 and in *Environmental Science and Technology* 2006; 40: 2531-36. Gauderman's group is well sponsored by a division of the California EPA. Imagine their funding stream if they reported no roadway effects?

Studies and analysis ignored by the EPA

The EPA also refuses to recognize the research and analysis that contradicts the EPA air regulation proposals.

Lipfert FW, Perry HM, Miller JP, et.al. The Washington University—EPRI veteran's cohort mortality study: preliminary results. *Inhal. Toxicol*. 2000, 12 S4:41-73. (Insignificant air pollution health effects.)

Enstrom J. Fine particulate air pollution and total mortality among elderly Californians, 1973-2002. *Inhalation Toxicology* 2005; 17:803-16. (Very large and long term study shows no air pollution death effect, in fact a counter intuitive protective effect of air pollution in many California cities. This study essentially nullifies the weak studies of Pope and Dockery as well as other death studies that are used by the EPA to push tighter NAAQS)

Moolgavkar S. Let. Fine particles and mortality. *Inhalation Toxicology 2006;18:93-4.* (Refutes the EPA air pollution project dogma. Discussion of EPA overreach and excessive regulatory zeal.)

Moolgavkar S. A review and critique of the EPA's rationale for a fine particle standard. *Reg Tox Pharm* 2005; 42:123-44. (Expose' of the EPA's failure to use good science to justify its agenda to make current ambient air pollution appear to be a serious health risk for Americans.)

Schwartz, J. *No Way Back: Why Air Pollution Will Continue to Decline*, (Washington: American Enterprise Institute, 2003). (Discussion of declining air pollution and improving air quality.)

The situation is so bad that the EPA and its sponsored epidemiologists and public health toxicologists control the literature and the journals. Journal editors now ignore toxicology and relative risk rule breaking. A recent poll by the National Institute for Statistical Science indicates that epidemiology journal editors no longer require data set production, p value calculation adjustments for multiple testing, and compliance with the rule on relative risk. The epidemiology journals have become political commentary on the hot environmental and social issues of the day—a mirror on the mental state of the academy.

Federal Rules of Evidence

The persistent failure of research on ozone and other air pollution observational studies to meet the requirement for relative risk of 2 and the p valued calculations without adjustment for multiple testing are examples of pseudo-science. The measure of scientific integrity, however, goes outside the academic and journal community. The Federal Courts have a stake in reliable evidence and the Federal Trial Court Judge makes the call on admissible scientific evidence.

The Federal Judicial Center's *Reference Manual on Scientific Evidence*, 2nd Edition (2000, West Group), also free on line at http://www.fjc.gov) Chapter on Epidemiology, written by highly esteemed experts, including Leon Gordis, the former Chair of epidemiology at Johns Hopkins School of Public Health deals with various matters of admissibility. The Manual states, at page 384:

> *The threshold for concluding that an agent was more likely than not the cause of an individual's disease is a relative risk greater than 2.0. Recall that a relative risk of 1.0 means that the agent has no effect on the incidence of disease. When the relative risk reaches 2.0, that implies that the agent is responsible (with certain qualifications noted below) and implies a 50% likelihood that an exposed individual's disease was caused by the agent.*
> *A relative risk greater than 2.0 would permit an inference that an individual plaintiff's disease was more likely than not caused by the implicated agent.*
> *Thus, a relative risk of 2.0 would permit an inference that an individual plaintiff's disease was more likely than not caused by the implicated agent.*

There are no major studies of ozone health effects relied on by the EPA that show a relative risk 2 or more. In fact there is not, at this time, a way to design a study on ozone that will show evidence of any relative risk, because there is no end point to measure. Ozone is a benign molecule, and doesn't cause death or disease. Exercise studies with excess exposure are a house of scientific cards for any EPA effort to build a toxicology argument against ozone.

The only reason the EPA can use these studies with relative risk of 1.5 or less, and not blush or apologize, is a political climate of panic about the environment and collusion in the academic and journal community collecting around the non-scientific social science concept of the precautionary principle. Discarding the relative risk rule is necessary to the survival of the precautionary principle, since the scientific evidence on ozone and most other pollutants cannot be shown to reach the relative risk of 2.

Expanding the effect of the EPA with "susceptibility."

The EPA also misuses the concept of sensitive or susceptible groups to make any exposure a concern for regulation. Susceptibility allows the EPA extraordinary latitude. There is always someone who is really, really sensitive—therefore the EPA plans to play the sensitive game and will make the society pay, eliminating any target toxin, regardless of the cost of the ablution. The rational regulatory regime does not adopt such a nonsensical approach, but the EPA embraces the concept as an excuse to overdo.

Reviewing the EPA United States air quality map, there are presently very few unsafe air quality areas, but that map will deceptively and dramatically change if the new ozone standard is implemented, along with the nonsense of the susceptibility. It will make no difference whether the standard is 0.06 ppm or .07 ppm, the non-compliance expansion guarantees that the EPA will exist into eternity.

The EPA is no longer in the business of protecting the public health and preserving the environment, the new range of ozone standards is an example of an EPA attempt to redefine what the environment should be and assure itself agency immortality. The EPA wants the world to be a scrubbed down bubble with no dust and no ozone for its own purposes, with no consideration of the rules of scientific integrity or even the mission of the agency to protect the environment and the public. Next the EPA will be regulating nitrogen, which is toxic if found at too high a percentage in the air. Really toxic.

The EPA is consciously and intentionally pushing the limits of scientific concepts of toxicity and epidemiology and cheating on the margins with the help of aggressive and flexible toxicology and epidemiology research. At this point a responsible Federal Judge, properly informed by the Federal Judicial Center *Reference Manual on Scientific Evidence*, chapters on toxicology and epidemiology, would throw out the "evidence" the EPA is using for this round of ozone standards.

The EPA refuses to study the health effects of the air quality improvements of the past 20 years. Why?

The EPA, like most government agencies or political advocacy groups, lives or dies by the old H.L. Mencken maxim about practical politics, that the public must be frightened, and anxious to be led to safety. False ozone fears and air pollution anxiety prop up the EPA. The EPA and its allies in the environmental movement feed the irrational and uninformed concern that the public has about a declining air quality, in the face of contrary evidence of improving air.

Why is there no research from the EPA that shows a public health benefit from the 20-year improvement in the quality of the air in the United States? Is the health benefit there and not shown or is it possible that the ambient air of 20 years ago, including the ozone levels, was not toxic? Generally even a blind toxicologist can prove a toxic effect by showing that the removal of a toxin caused a benefit. If there are air quality improvements that the EPA documents in its monitor information, then there should be a corresponding improvement in the health of the public.

Los Angeles and Houston air have improved—why no research to show the benefits? Is the EPA a one trick pony—they can only talk panic and crisis and bad air. Good air is not in the lexicon, only bad air and assertions of people dying from bad air? The proof of

benefit would be the logical scientific inquiry to show the value of EPA activity and tighter air standards. Where are those studies of benefit?

If there is no real change in life expectancy or quality of life from air quality improvements, what will the EPA do, more importantly what should the country and the society do? Fire the EPA for lying or malpractice? The EPA and its allies in state and local government agencies, and in the non-governmental environmental advocacy sector would be decimated by reports that there is no crisis in the environment, never was. They would also be, incidentally, unemployed and unemployable as pollution sheriffs.

Air Pollution Trends and Policy

Some places in America will be naturally dusty; some places will have natural background ozone levels that create haze. West Texas exemplifies the first, the Smoky Mountains the second. Trends in air pollution, control of ozone and ozone precursors in the past 30 years have all been positive, yet the EPA does not and will not report any benefit or improvement and continues to aggressively and energetically pursue every opportunity to increase its regulatory empire and authority. The EPA even sponsors and funds non-governmental entities like the American Lung Association and other rabid environmental groups that sue the EPA to push more environmental intrusions. That raises a question about conflicts and influence peddling, and contaminates the very important debate about EPA responsibilities to protect health and preserve the environment and maintain a high level of integrity in its science and research.

The blow back on the latest round of EPA overreach in ramming down the ozone standard is the protest of reasonable people confronting a new regulatory burden based on weak science. Ten years ago the EPA Clean Air Scientific Advisory Committee advised the EPA that ozone could not be shown to produce adverse health effects at the standard then, 0.12 PPM. Even then the CASAC, which is inclined to favor EPA policy proposals as a creature of the agency, was reluctant to support the ozone standard reduction from 0.120 ppm to a lower number. Chairman George Wolff said " although the panel member's opinions differed, none supported the lower end of EPA staff's recommendations, and a majority of members stated a position which included . . . the present standard."

EPA Clean Air Scientific Advisory Committee

The EPA Clean Air Scientific Advisory Committee (CASAC) in the late 1980s pointed out that ozone respiratory effects were not "adverse" health effects, and the CASAC in the 1990s refused to support using the Pope and Dockery studies to justify new NAAQS in 1997, but now the EPA is less scientific or objective in its analyses. The CASAC of today has become an advocacy committee committed to EPA agendas, even advocating more aggressive EPA activity. The CASAC of today has not and cannot be objective

about ozone issues, and the current CASAC commentaries are not objective science but advocacy for aggressive environmentalism, now and forever.

There is no explanation for the CASAC conduct of the past few years other than political commitment to the environmental movement and the precautionary principle. In the past the CASAC and other agencies were the only chance that fanatic EPA officials would be brought under control, but now the CASAC has gone to the political side and cannot be trusted to show objectivity. Any argument for more regulation is supported. They represent the politicization of environmental science. CASAC commentary on small particulates last year was over the top.

Only 6 of 21 CASAC members supported the small particulate standards in 1996, the CASAC in 1996 advised in favor of the standard for ozone remaining at 0.120 ppm. Times have changed, the CASAC is now no restraint on junk science, and the CASAC of today is predictably in favor of any new and more stringent standard.

There are many in America who believe that the air quality is worse now than ever. That is because they get no reliable information from the EPA. The EPA is no longer a public agency that protects the public, but a political propaganda mill, intent on panicking the public and working an environmentalist agenda. Informing the public of the improvements in air quality would reduce public anxiety and EPA and environmental group funding. Environmentalism would suffer a setback as a movement. The EPA is intentionally giving the public incorrect information about the current air quality, creating more anxiety, pollution warnings and claims about deaths.

This proposed new ozone standard is part of the deception, since the day the standard goes into place the American Lung Association, the EPA and the usual environmentalist organizations like Sierra Club will announce a new dirty air crisis. This latest round for ozone standard setting appears to be an effort by an EPA and its allies to reinvigorate their position as protecting the innocent public from killer air. They offer the naïve members of he public the proposal to create a pristine environment, more pristine than even Mother Nature could produce.

Consider, instead the reality as described by an environmental regulation expert:

> *The United States has made tremendous progress in reducing air pollution during the last forty years. Air pollution has declined dramatically since the 1960s and 1970s, and virtually the entire nation now meets federal health standards for carbon monoxide (CO), sulfur dioxide (SO_2), and nitrogen dioxide (NO_2). Many areas of the country still exceed health standards for ground-level ozone ("smog") and airborne particulate matter (PM), but both of these pollutants continue to decline as well. Half of the nation's ozone-monitoring locations exceeded the federal one-hour ozone standard in the early 1980s, but only 13 percent exceeded the standard by the end of 2002. PM measurement methods have changed a number of times during the last forty years, but all trend data show PM levels dropping. Average levels of $PM_{2.5}$—the form of PM now of*

> *greatest regulatory concern—have declined by a third during the last twenty years.* (Joel Schwartz, 2003)

A good example of irrational panic mongering is in the September 9, 2004 issue of *New England Journal of Medicine,* in which C. Arden Pope, an economist *cum* environmentalist, describes as a companion piece to another children are victims of bad air article, describes killer air in Belgium in 1930, Pennsylvania in 1948, and London in 1952 and proposes those incidents as examples of why he thinks there is good reason to be worried. Pope is always worried, although he can't show me one person in his studies who really died from air pollution. They died as members of the cohort and he counted them as dead from air pollution after he looked at their death certificates. That's not a proper toxicologic analysis, that's an association. People don't die on epidemiologist's desks from associations.

In America ambient air pollution did not kill anyone, last week, last year, or in the last ten years. The crisis of bad air is long past, and the real health effects from air are non existent, but won't go away because the EPA is too big and too influential and too aggressive to go silent.

I agree with the Chairman of the Texas Commission on Environmental Quality, Buddy Garcia, who said in his letter of September 25, 2007 to EPA Administrator Johnson that ozone non-compliance will be the rule rather than the current exception, if the new standard is put in place. Mr. Garcia points out that 0.06 is well known to the EPA as a background level in many environments—and that such a standard is irrational and cannot be complied with in places like the Gulf Coastal Plain.

Chairman Garcia also points out a little problem that the EPA ignores, that ozone precursors are mostly a product of mobile sources, not point/stationary sources, so the penalties and costs will be imposed on cities and communities for things they can't fix. Why is it that the EPA appears to care little about Mr. Garcia's concerns and his appeals for sensible science and policy making?

Summary

The research used to justify the proposed new ozone standard does not demonstrate results that meet the basic rule for proof of detrimental health effects. In fact the consistent findings of the EPA ozone research is insignificant ambient ozone pollution relative risk and laboratory evidence of fleeting effects if humans or animals are forced to breath high levels of ozone and exercise.

Research studies have shown that low relative risk results and pervasive confounders make it very unlikely that the proposed new ozone rules will have measurable beneficial or protective health effects. The EPA has failed to show the previous reduction in ozone levels has produced any benefits.

The EPA should abandon this precautionary-principle driven and junk science justified new standard, and retreat from continued aggressive tightening of ozone and other air quality standards.

Conclusion and recommendation.

There is no health effects science that justifies the current ozone standard of 0.08 ppm, so I urge the EPA to reset the ozone standard at the more reasonable 0.12 ppm, pending evaluation of the ozone control program for termination. Ozone should go the way of large particles, no longer on the list of EPA targets.

Imagine a government control program that has an end.

Economic and political effects of adoption of the recommendation.

I project that billions of taxpayer dollars and compliance costs could be returned to the citizens as soon as the EPA gives up chasing ozone, a benign component of the natural world.

I also project that a chastened and re-dedicated EPA might, after the end of the ozone campaign, eschew future goose chases, and focus on serious, non-political, scientific inquiries in the public interest.

Contact information

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Appendix E

Here is a multi-year California study by biostatistician and attorney Steve Milloy that shows no mortality effect from small particle air pollution. All but the most remote and uninhabited counties with no air monitors were included.

http://junksciencecom.files.wordpress.com/2013/12/california-pm25-experience-2007-2010-final.pdf

Here is Milloy's multi year study that shows no ozone effect on asthma at UC Davis Hospital outside Sacramento.

http://junkscience.com/2013/09/03/study-ozone-not-linked-with-asthma-hospitalizations-in-major-california-hospital-system/

Appendix F

Review and Critique of the Environmental Protection Agency's Analysis and Conclusions Regarding the Effect of Climate Change on Future Ozone Levels and Ozone-Related Health Effects

Joel Schwartz

Consultant

June 19, 2012

Introduction

This report presents a review and critique of EPA's analysis and conclusions regarding the effects of climate change on future ozone levels (EPA 2009c; EPA 2009a; EPA 2009b). Our findings can be summarized as follows:

With or Without Climate Warming, Ozone Will Decline Substantially in the Future. The lesson of the past few decades is "higher temperatures, lower ozone." Ozone declined all over the U.S., with the greatest improvements occurring in the most polluted areas of the country. The ozone declines were due to reductions in ozone-precursor emissions. Already-adopted measures will eliminate the vast majority of remaining ozone-precursor emissions during the next few decades, resulting in continued ozone reductions, even if the climate warms in the future.

EPA's ozone modeling exaggerates both future ozone levels and the absolute effect of climate change on future ozone levels. The studies EPA relies on for predictions of future ozone levels generally use ozone-precursor emission inventories from somewhere between 1996 and 2001 to "predict" ozone levels in the 2050s or 2090s. These studies are trying to predict the effect of warming on ozone levels in the 2050s using an ozone-precursor emissions inventory that is twice as high as *current* emissions and about ten times greater than likely ozone-precursor emissions in the 2050s. The use of such unrealistic scenarios renders EPA's ozone modeling invalid as a guide to the effects of climate warming on future ozone levels.

Both modeling and observations suggest that ozone-precursor reductions between the late 1990s and 2011 have already eliminated most of the "climate penalty". Since ozone-precursor emissions are dropping rapidly, whatever climate penalty remains will likely disappear within a decade or two, as most remaining ozone-precursor emissions are eliminated by already-adopted measures.

EPA implies that the Clean Air Act requires EPA and states to allow ozone to increase to dangerous levels before they can take action to reduce ozone, and that this means warming has to cause harm before EPA can do anything about it. While this may, in principle, be the legal structure of the Clean Air Act, it has little to do with the reality of how measures to reduce ozone precursors have been and are implemented. EPA has been pre-emptively reducing ozone-precursor emissions for decades in all areas of the U.S. and will continue to do so. Local and state governments are taking additional actions to reduce the few sources of ozone precursors that are not under federal control. There has not been, is not now, nor will there be in the future any location in the country that is just sitting around waiting for ozone levels to rise.

Ozone at current, historically low levels is not a significant human health concern, and future ozone levels will be much lower, regardless of climate warming. EPA's claims for ozone's most serious health effects—premature death and respiratory and cardiovascular hospitalizations—are based on the results of

observational epidemiology studies. However, in cases where observational studies have been tested against randomized controlled trials, the observational study results are nearly always falsified.

Laboratory studies with several different animal species show that animals do not die, even when exposed for the equivalent of many years to ozone at levels nearly ten times greater than the highest ambient levels. These results make it biologically implausible that ozone at real-world outdoor levels could be causing premature death in humans.

Studies of low-level ozone exposure with human volunteers demonstrate that an 8-hour standard of 85 ppb is more than stringent enough to protect human health with an adequate margin of safety, even from the most mild health effects EPA claims for ozone.

In its characterization of ozone's health effects, EPA selectively emphasizes studies and portions of studies reporting harmful ozone effects, while downplaying studies reporting no effects or apparently protective ozone effects. This creates an impression that the evidence for harm from ozone at real-world levels is far more robust and consistent than the full weight of the evidence suggests.

The overall result of the above considerations is that the 85 ppb 8-hour ozone standard protects human health with plenty of room to spare. Peak annual ozone levels are already below this level in about 90 percent of the country. Continued ozone-precursor reductions will ensure that the entire country will be well below this level by the time any significant additional warming occurs. Thus, even if warming causes ozone to decrease a bit less than it otherwise would have, future ozone levels around the U.S. will still be below a level of concern for human health, with or without climate warming.

Taken together the weight of the evidence indicates that EPA has exaggerated both future ozone levels and the effects of warming on ozone levels. In reality, future ozone levels will be below a level of concern for human health, regardless of whether the climate warms.

1 With or Without Climate Warming, Ozone Will Decline Substantially in the Future

Even if EPA is correct about how much the climate will warm and the effect of warming on ozone levels, the worst-case scenario in a warming climate is the following: ozone will decrease substantially during the 21st Century with or without climate warming, however, ozone will decrease slightly less substantially if the climate warms as much as EPA claims it will.

We can draw this conclusion for the following reasons: (i) During the last few decades this is exactly what has happened. The U.S. climate warmed about 2°F, but ozone dramatically declined. Areas with the worst ozone achieved the greatest improvements. Ozone declined because ozone-precursor emissions declined.

(ii) The fact that peak ozone levels and ozone exceedance days have dropped even as the climate has warmed, shows that ozone formation is becoming less and less sensitive to temperature. (iii) Ozone-precursor emissions will continue to dramatically decline. Just as in the past, the result will be continued declines in ozone, even with warming. To the extent any significant warming occurs in the next few decades, nearly all anthropogenic ozone-precursor emissions will have been eliminated, mitigating concerns over the effect of warming on ozone levels.

Between 1970 and the present, the U.S. climate warmed about 2°F, yet ozone dramatically declined. The lesson of past experience is "higher temperatures, lower air pollution."

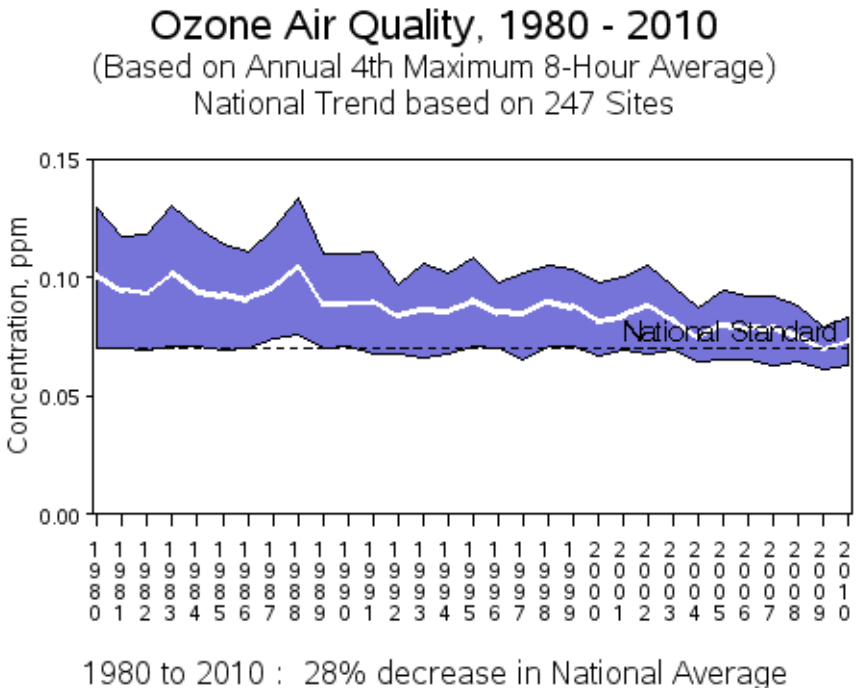
Past experience shows that reducing ozone-precursor emissions reduce ozone, regardless of whether the climate warms. EPA itself reports dramatic declines in ozone during the last few decades, despite climate warming over the same period. EPA's Technical Support Document (TSD) for its Endangerment Finding states, "According to studies cited in Karl et al. (2009), the annual average temperature in the Northeast has increased by 2°F (1°C) (relative to a 1960-1979 base period) since 1970" (EPA 2009c). Nevertheless, EPA's own monitoring data demonstrate that ozone levels decreased dramatically during the same period, as shown in Figure 1. Figure 1 comes directly from EPA's own AirTrends website (http://www.epa.gov/airtrends/ozone.html, accessed June 10, 2012).

As the graph shows, from 1980-2010, average peak ozone levels decreased by 28 percent. Peak ozone levels improved even more in areas with the highest ozone levels. The top of the blue-shaded area represents the 90th percentile among all monitoring locations in the U.S. Note that the 90th percentile ozone level declined from about 125 ppb in 1980 down to about 80 ppb in 2010, a 36 percent decrease.

Figures 2 and 3 show similar data over a longer time period. Figure 2 shows that average peak annual 8-hour ozone declined more than 30 percent from 1975 to 2010, while the maximum 8-hour ozone level declined more than 65 percent. Not only did peak levels of ozone decline, but Summer-average ozone levels declined also. Figure 3 shows that June-August average ozone declined as well. At the average monitoring location, June-August average ozone declined about 18 percent from 1975-2010, while June-August average ozone at the worst location in the country declined about 35 percent.

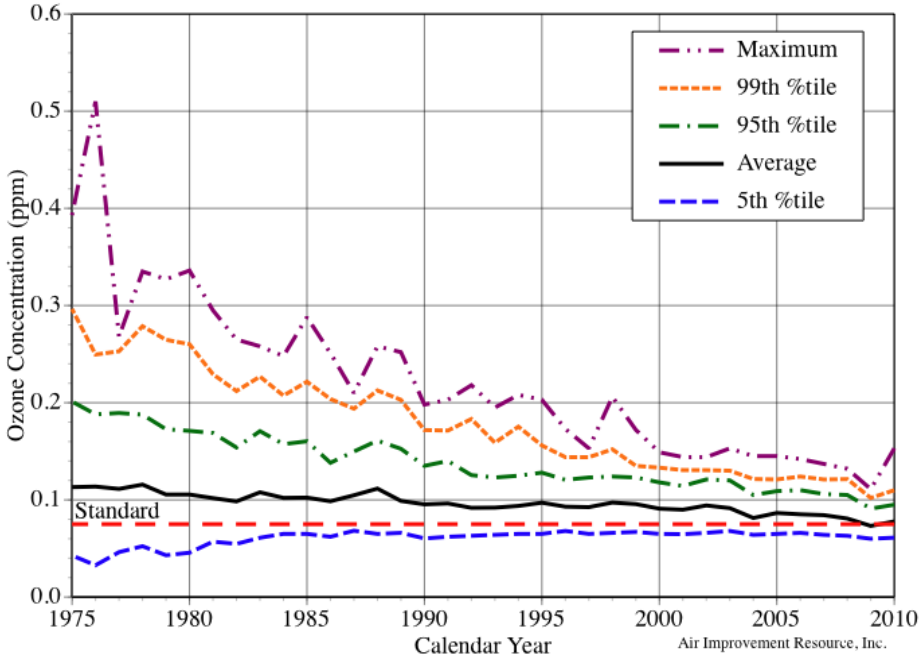
Overall, the lesson of the last 40 years is "higher temperatures, lower ozone." It is, of course, possible that ozone would have been even lower had the temperature not warmed. Regardless, the fact is that 2°F of warming did not prevent dramatic declines in ozone levels during the last 40 years. EPA never explains why we should expect the future to be the opposite of the past and does not even mention that past ozone levels declined dramatically despite warming of similar magnitude to what it predicts will occur between now and 2050.

Figure 1. National Trend in Peak Ozone Levels from 1980-2010



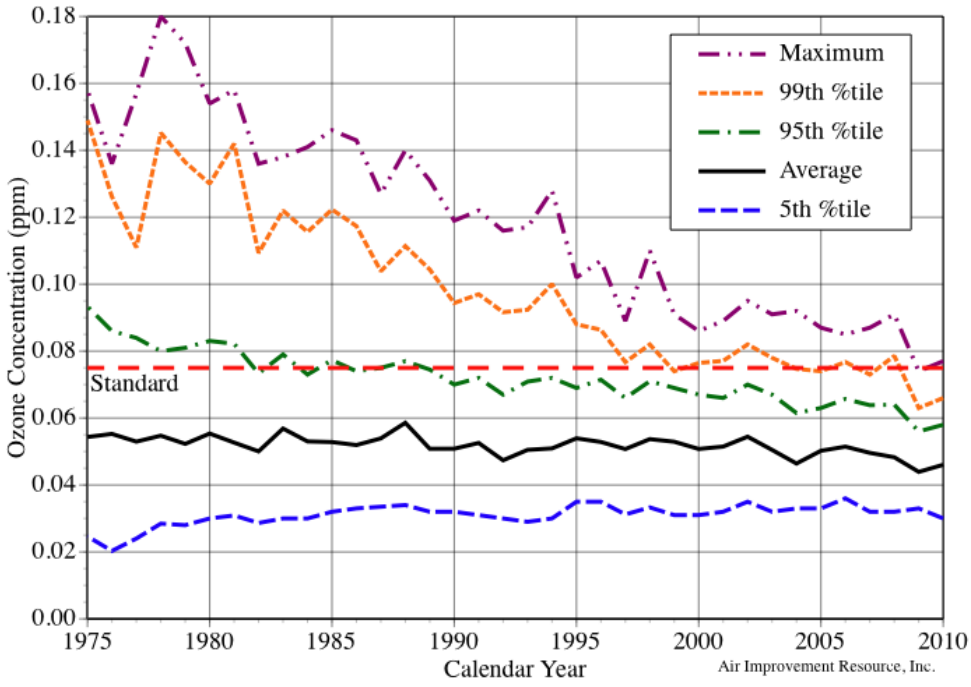
Source: Graphic downloaded from http://www.epa.gov/airtrends/ozone.html (accessed June 12, 2012).

Figure 2. Highest Annual 8-hour Ozone Concentrations for All U.S. Monitoring Locations from 1975-2010



Source: Dennis Kahlbaum, Air Improvement Resource, using ozone monitoring data downloaded from EPA.

Figure 3. June-August Average of Daily Peak 8-hour Ozone Concentrations for All U.S. Monitoring Locations from 1975-2010



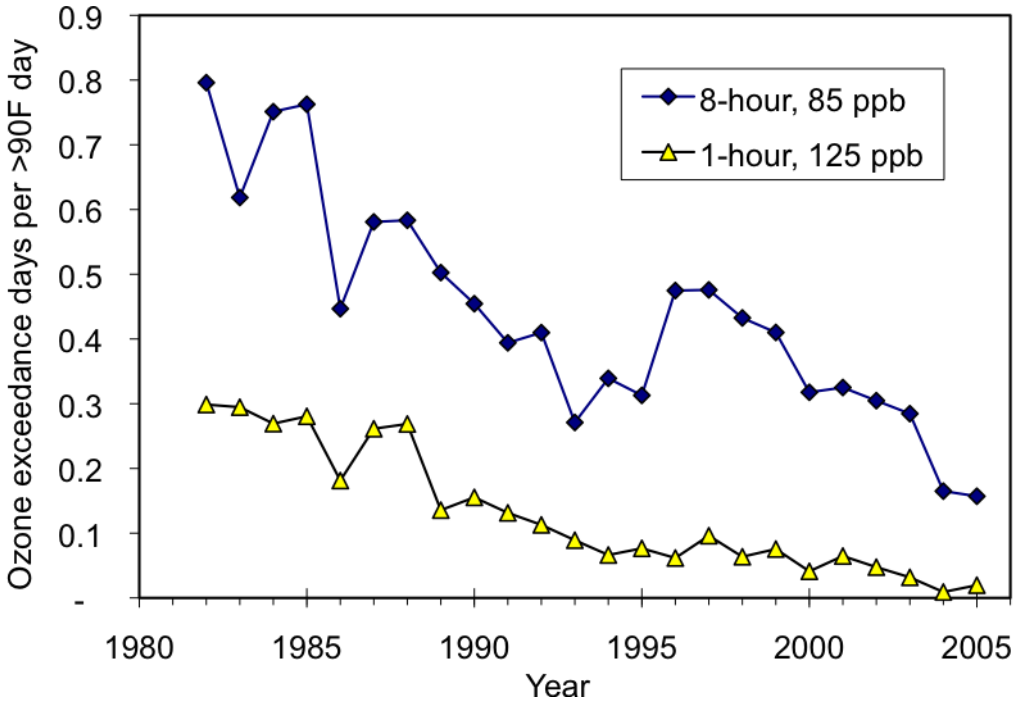
Source: Dennis Kahlbaum, Air Improvement Resource, using ozone monitoring data downloaded from EPA.

The fact that ozone has declined as the climate has warmed suggests that ozone levels are becoming less and less sensitive to temperature over time.

The fact that ozone has declined as the climate has warmed suggests that ozone levels are becoming less and less sensitive to temperature over time. One way to check this is by looking at the ratio of the number of ozone exceedance days each year to the number of hot days each year in a given city. Schwartz and Hayward (2008) did such an assessment for a number of U.S. cities representing major geographical areas of country and including cities with the highest ozone levels in the nation. The results are shown in Figure 4.

Figure 4 shows the ratio of the number of days exceeding a given ozone level to the number of days exceeding 90ºF each year average over 10 cities. In the early 1980s, the number of 8-hour, 85 ppb ozone exceedances per hot day was around 0.6 to 0.8. By the mid-2000s, the ratio had dropped to about 0.15 to 0.3. The improvement was even more dramatic for the higher ozone levels probed by the old 125 ppb, 1-hour standard. Between 1982 and 2005, the number of 1-hour exceedance days per hot day dropped from 0.3 to near zero. In other words, ozone levels have been becoming steadily less and less sensitive to temperature. The fact that ozone levels have continued to decline in the years since 2005 shows that this downward trend in the sensitivity of ozone levels to temperature has continued.

Figure 4. Trend in the Ratio of Days per Year Exceeding A Given Ozone Level to Days per Year With Temperature Greater Than 90°F



OURCES: Air pollution data were downloaded from EPA's Air Quality System (AQS) database, http://www.epa. gov/ttn/airs/airsaqs/detaildata/downloadaqsdata.htm and http://www.epa.gov/ttn/airs/airsaqs/archived% 20data/downloadaqsdata-o.htm (accessed November 27, 2006). Temperature data were downloaded from the National Climatic Data Center, Summary of the Day (Data Set TD-3200), http://ncdc.noaa.gov (accessed October 3, 2006).

NOTES: Figure is based on ozone and temperature data for ten metropolitan areas: Atlanta, Baltimore, Charlotte, Chicago, Cincinnati, Houston, Los Angeles, Nashville, New York, and Philadelphia. Ozone exceedance days for a given metropolitan area were calculated as the average number of exceedance days each year for all monitoring sites in an area with continuous data. This was then divided by the number of days each year with peak temperature greater than 90°F. The graph gives the average ratio across the ten metropolitan areas. The year 1982 was the earliest time period for which all of the cities had at least one continuously operated monitoring site.

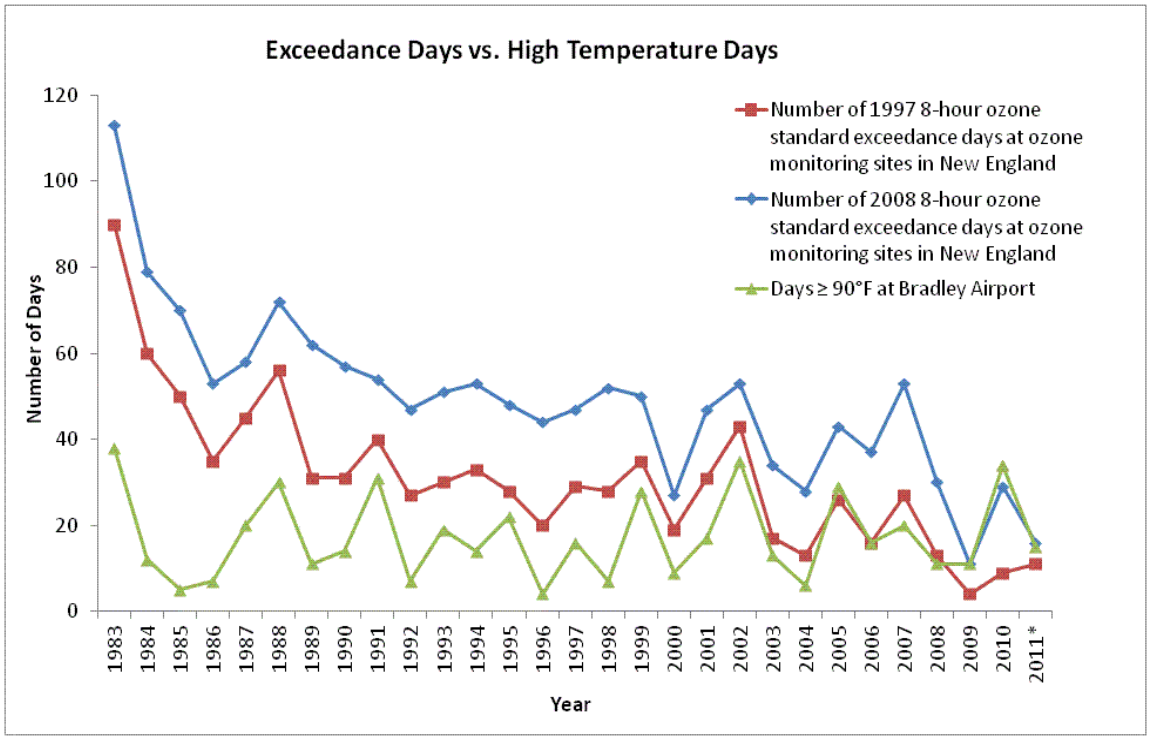
Indeed, EPA itself presents data showing that ozone is becoming less and less sensitive to temperature. Figure 5 was downloaded from the EPA Region 1 (New England) web site. It shows the number of ozone exceedance days in New England from 1983 to 2011 based on both the 85 ppb 8-hour ozone standard and the new 75 ppb 8-hour ozone standard.

First, note that the number of ozone exceedance days has substantially declined. Exceedances of the 85 ppb threshold decreased from about 40 to 50 days per year in the late 1980s down to about 5 to 10 days per year during the last few years.

Likewise, exceedances of the 75 ppb threshold dropped from about 55 to 70 days per year down to about 15 to 30 days per year over the same period.

Second, note that ozone levels are becoming much less sensitive to temperature. The graph shows that 1983, 2002, and 2010 all had about 39 days of at least 90°F, yet the number of 75 ppb ozone exceedance days dropped from 115 to 55 to 30 for the three time periods, respectively. Likewise, the number of 85 ppb exceedance days dropped from 90 to 43 to 10, respectively. In other words, given similarly hot summers, the number of 75 ppb and 85 ppb ozone exceedance days dropped 74 percent and 89 percent, respectively. And this is in the Northeast—the region EPA says is among the most sensitive to the effects of temperature on ozone. Once again, this suggests that temperature is a minor factor when compared with reductions in ozone precursors.

Figure 5. Trend in Number of Ozone Exceedance Days vs. High Temperature Days in the New England Region.



Source: EPA Region 1 (New England) web site: http://www.epa.gov/region1/airquality/graph.html (accessed June 13, 2012).

Note: Over time not only has the number of ozone exceedance days declined, the sensitivity of ozone to temperature has also declined. For example, in New England, since the early 1980s, the number of 75 ppb or 85 ppb ozone exceedance days during hot summers has dropped by 74 percent and 89 percent, respectively.

Recent studies also suggest that reductions in ozone precursors make ozone levels less sensitive to temperature. For example, Bloomer et al. (2009) concluded, based on observations of the real-world response of ozone to NOx reductions, that a 43 percent reduction in NOx emissions from power plants reduced the "climate

penalty"—the amount by which ozone increases per degree of increase in temperature—by 31 percent, from 3.2 ppb ozone/°C to 2.2 ppb ozone/°C.

It is extraordinary that in hundreds of pages of documents claiming to have performed a comprehensive scientific analysis of future ozone levels in a warming climate, not once does EPA mention the simple fact that ozone has dramatically declined as the climate has warmed, or discuss the implications of this observation for predicting the effect of warming on future ozone levels.

Ozone will continue to decline in the future, with or without climate warming, because EPA will eliminate most remaining ozone-precursor emissions during the next few decades

EPA's TSD for its Endangerment Finding states, "Temperatures in the Northeast are projected to rise an additional 1.4 to 3.4°F (0.78 to 1.9°C) in summer over the next several decades (across low and high emissions scenarios)" (EPA 2009c). In other words, the temperature rise of 2ºF that EPA estimates for the last four decades is in the same ballpark as the range of temperature increases (1.4 to 3.4 ºF) that EPA predicts for the next several decades. Thus, as long as ozone-precursor emissions continue to decline, we can expect that ozone will continue to decline as well, regardless of any climate warming.

Ozone precursors will indeed continue to decline. In fact, over time EPA has only intensified its efforts to reduce ozone-precursor emissions, because its progressively tighter ozone standards can't be attained without large reductions in ozone precursors. As we show below, the measures EPA already has in place will progressively eliminate nearly all remaining ozone-precursor emissions during the next few decades. EPA shows no signs of reducing its production of new emissions control regulations and will no doubt adopt additional ozone-precursor reduction measures in coming years. Indeed, among other new regulations currently in the planning stages is a "Tier 3" rule to require additional emission reductions from automobiles.[1]

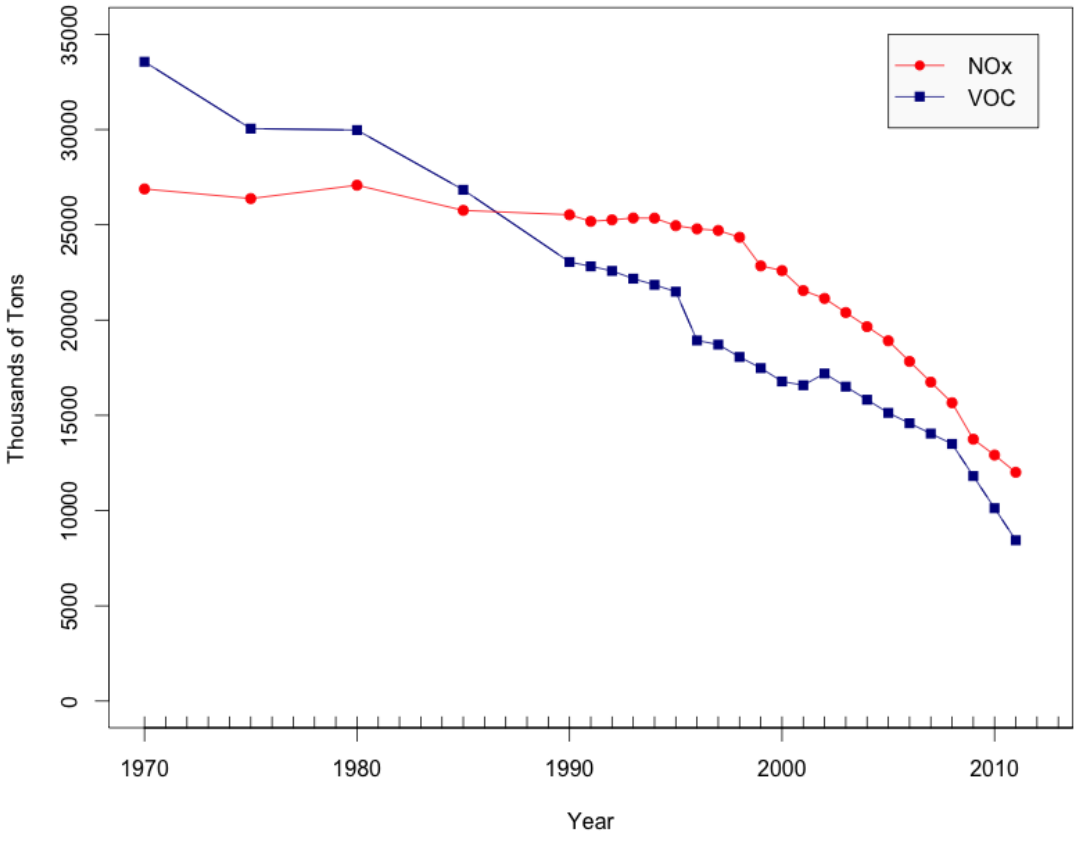
Figure 6 displays the national trend in emissions of ozone precursors from 1970 to 2011 (EPA 2012a). Note that the rate of reduction in both NOx and VOC emissions has been accelerating in recent years. Large annual reductions in NOx and VOC emissions will continue, even if EPA adopts no new regulations to control emissions, because most of the emissions reductions from existing regulations have not yet been achieved. For example:

In 2004, EPA implemented Tier 2 standards for automobiles (cars, SUVs, pickup trucks, and minivans), which reduced NOx, CO, and VOC emissions by 77 to 95 percent (with the largest percentage reductions apply to the highest-emitting vehicle classes) when compared with the Tier 1 standards that were implemented in 1994.[2] In 2007 EPA implemented new standards for on-road heavy-duty diesel

1 See http://www.epa.gov/rfa/tier3.html. Unless otherwise noted, all web links provided in this document were current as of June 16, 2012.
2 http://www.epa.gov/tier2/

trucks that reduced emissions from those vehicles by 90 percent below previous requirements.[3] In 2010, EPA implemented Tier 4 standards for non-road heavy-duty diesel vehicles (e.g., construction and farm equipment), which reduced emissions from those vehicles by 90 percent below previous requirements.[4] EPA has recently implemented requirements for many other types of mobile sources, such as trains, marine engines, snowmobiles, mowers, and many other non-road emissions sources.[5] Taken together, even after accounting for population growth, these requirements will progressively eliminate more than 80 percent of ozone-precursor emissions from mobile sources during the next few decades, as the fleet turns over to models meeting these standards.

Figure 6. U.S. Anthropogenic Emissions of NOx and VOC from 1970 to 2011



Source: EPA, "1970 - 2011 Average annual emissions, all criteria pollutants," http://www.epa.gov/ttnchie1/trends/ (accessed June 13, 2012).

Note: NOx and VOC emissions have been declining rapidly. The rate of decline has been accelerating in recent years.

EPA has likewise implemented dozens of rules for stationary and area pollution sources, many of which have future compliance dates. For example, the Clean Air

3 http://www.epa.gov/otaq/hd-hwy.htm
4 http://www.epa.gov/nonroad-diesel/.
5 http://www.epa.gov/nonroad/

Interstate Rule requires NOx and SO2 from power plants to be reduced by 70 percent.[6] Eventually, the Cross State Air Pollution Rule (CSAPR) or some successor rule will require additional reductions from power plants.[7] Dozens of Maximum Achievable Control Technology (MACT) rules, require emissions reductions from nearly all industrial sources.[8] EPA's New Source Performance Standards (NSPS) require all new industrial construction to meet either the Lowest Achievable Emission Rate (LAER) or Best Available Control Technology (BACT) when constructing a new industrial facility. Thus, capital stock turnover during the next few decades will likewise continue to eliminate air pollutant emissions as old facilities are retired and new facilities with state-of-the-art pollution controls are constructed.

EPA will also continue to adopt new rules. A "Tier 3" standard for automobiles is already in the planning stages.[9] More rules will be coming as EPA clamps down further on what few emissions remain in order to help states attain EPA's progressively tighter ozone and particulate matter standards.[10]

Despite the certainty of continued large emission reductions during the next few decades, EPA in its TSD tries to create a false impression of uncertainty about future air pollutant emissions, stating: "Further, the range of plausible short- lived emission projections is very large. For example, emission projections used in CCSP (2008d) and in the IPCC *Fourth Assessment Report* (IPCC, 2007a) differ on whether black carbon particle and nitrogen oxides emission trends continue to increase or decrease. Improvements in our ability to project social, economic, and technological developments affecting future emissions are needed" (TSD, pp. 92-93). It may be true that the Climate Change Science Program (CCSP) and the IPCC chose to assume higher air pollutant emissions in the future. But this is a separate issue from whether such assumptions are plausible. Clearly they are not. As EPA knows full well, the requirements it has already adopted will eliminate nearly all remaining ozone-precursor emissions and it has more rules in the planning stages.

Although ozone-precursor emissions will be far lower on in coming decades, it is difficult to predict exactly how much lower. Nevertheless, it is clear that EPA has already adopted requirements sufficient to eliminate at least 80 percent of remaining ozone-precursor emissions, even after accounting for economic and population growth. Furthermore, both EPA's emissions inventory trend (see Figure 6, above) and studies of trends emissions and ambient levels of ozone precursors are consistent with these conclusions.[11] In other words, current ozone-precursor emissions are about five times greater than ozone-precursor emissions are likely to

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6 See http://www.epa.gov/cair/
7 See http://www.epa.gov/airtransport/
8 See http://www.epa.gov/ttn/atw/mactfnlalph.html
9 See http://www.epa.gov/rfa/tier3.html
10 EPA adopted a 75 ppb ozone standard in 2008 and just announced a tougher $PM_{2.5}$ standard.
11 See, for example, Bishop and Stedman (2008).

be in 2050, while mid-to-late 1990s ozone-precursor emissions were about ten times greater than likely emissions in 2050. It is this reality, not the errant assumptions of the CCSP or the IPCC, that EPA should have used as the basis for its TSD.

2 The studies that EPA relies on for predictions of future ozone levels use unrealistically high emissions of ozone precursors, which exaggerates both future ozone levels and the absolute effect of climate change on future ozone levels.

The studies EPA relies on for predictions of future ozone levels generally use ozone-precursor emission inventories from somewhere between 1996 and 2001 to "predict" ozone levels in the 2050s or 2090s. However, even *current* ozone-precursor emissions are already only half of levels during the late 1990s and dropping rapidly. Also, as shown in Section 1, as a result of already-adopted and soon-to-be-adopted measures, U.S. ozone-precursor emissions in the 2050s will likely be no more than about one-tenth of late 1990s levels.

This means studies that use late 1990s ozone-precursor emissions and apply them to the predicted climate of 2050 are using an ozone-precursor emission inventory that is already twice as high as *current* ozone-precursor emissions and about *ten times greater* than a realistic ozone-precursor emissions inventory for 2050. As a result, both ozone levels and the absolute effect of warming on ozone levels will be far lower in the future than suggested either by EPA or the peer-reviewed research literature in general.

The studies EPA relies on to "predict" the effect of climate warming on ozone use drastically inflated ozone-precursor emissions inventories

EPA bases the ozone results of its Endangerment Finding (EF) on its report "Assessment of the Impacts of Global Change on Regional U.S. Air Quality: A synthesis of climate change impacts on ground-level ozone" (EPA 2009a), which it refers to as the "Interim Assessment" (IA) in its Endangerment Finding. As EPA states in the IA, "The aim of this phase was to consider the effects of climate change in isolation, without accompanying changes in anthropogenic emissions of precursor pollutants expected to occur over the same timeframe." (IA, p. xxii).

Table 1 displays assumptions about future ozone-precursor emissions in studies EPA cites in its Endangerment Finding. Table 2 provides similar information for studies cited in EPA's Interim Analysis. Reading from the left, the first column gives the citation. Articles from the same research group using the same assumptions are grouped together. The second column lists the "base year" for the ozone-precursor emissions inventory. The modeling was done either with ozone-precursor emissions equal to the emissions during this base year or scaled by some factor relative to this base year. The third column lists emissions of NOx and VOC assumed for 2050, relative to the base year from the previous column. For example, "no change" means

that emissions were assumed to be the same in 2050 as in the base year; +29% means emissions were assumed to have risen 29 percent between the base year and 2050. Most of these studies used the highest-warming IPCC scenarios (A1 or A2) to project future climate conditions, so their results are worst-case scenarios for the effect of climate warming on ozone levels.

Table 1. Assumptions About Future Ozone-Precursor Emissions in Studies of the Effect of Climate Change on Future Ozone Levels: Studies Cited by EPA in its Endangerment Finding

Article	Emissions Inventory Base Year	2050 Emissions, Relative to Base Year		Notes
		NOx	VOC	
Bell et al. (2007), Knowlton et al. (2004), Hogrefe et al. (2004)	1996	No Change		No change for the base scenario. Increased emissions for sensitivity analysis.
		+29%	+8%	
Denman et al.	1996	No Change		This is the IPCC AR4 report. EPA cites it, and it cites Knowlton et al. (2004) for future ozone levels.
Hauglustaine et al. (2005)	2000	+77%	+85%	Inventory change is for OECD countries. U.S.-only inventory not provided. Simulation for 2100, not 2050.
Liao and Seinfeld (2006)	2000	+77%	+85%	Inventory change is for OECD countries. U.S.-only inventory not provided. Simulation for 2100, not 2050.
Jacob and Winner (2009)	Review article			
Mickley et al. (2004)	2000	No Change		Not an ozone study, but looked at the effect of warming on black carbon and CO levels.

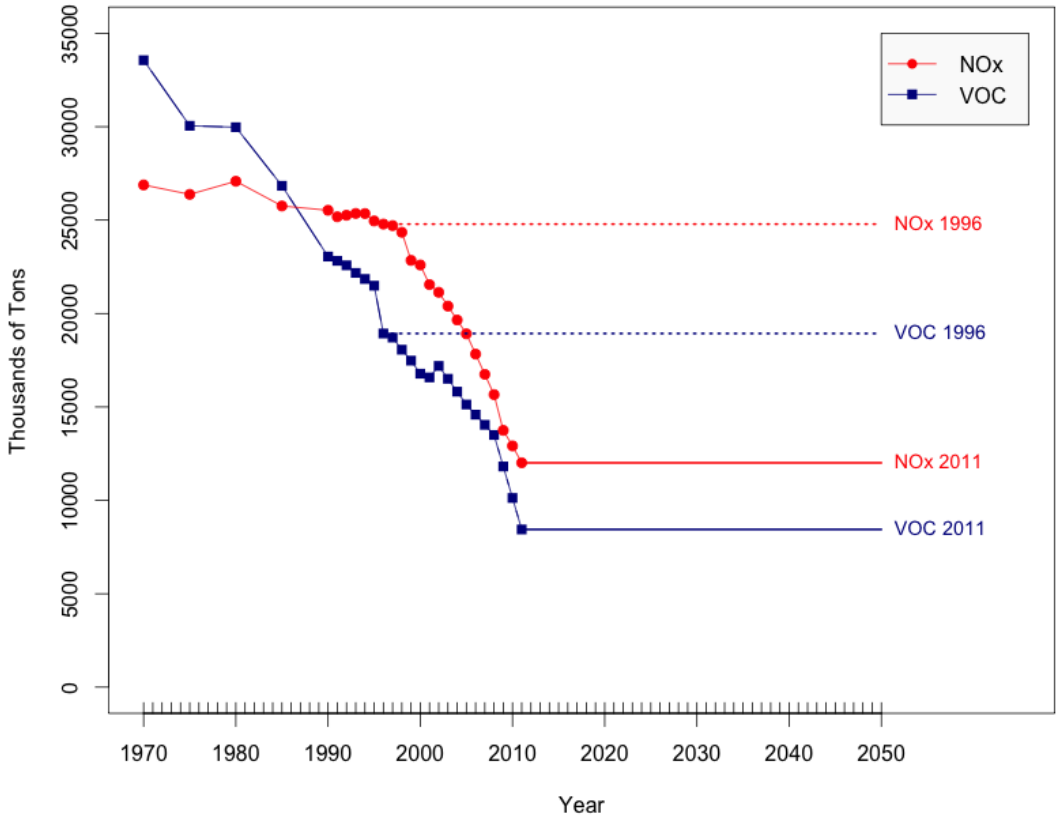
Table 2. Assumptions About Future Ozone-Precursor Emissions in Studies of the Effect of Climate Change on Future Ozone Levels: Studies Cited by EPA in its Interim Analysis

Article	Emissions Inventory Base Year	2050 Emissions, Relative to Base Year		Notes
		NOx	VOC	
Avise et al. (2009), Chen et al. (2009)	1999	+6%	+50%	Included growth in emissions due to economic and population growth, but excluded reductions in emissions due to regulations.
Aw and Kleeman (2003)	1995-1997	No Change		
Dawson et al. (2009)	2001	No Change		
Murazaki and Hess (2006)	1997	No Change		Simulation for 2100, not 2050
Racherla and Adams (2008)	1990s	No Change		
Racherla and Adams (2006)	1990s	No Change		
Steiner et al. (2006)	2000	NOx -10% to -50%		Emission reduction percentage varied by region of California.
		VOC -50% to -70%		
Tagaris et al. (2008), Liao et al. (2008), Woo et al. (2008)	2001	-55%	-40%	
Tao et al. (2007)	1999	+30%	~+5%	A1FI Scenario
		-50%	-10%	B1 Scenario
Wu et al. (2008a), Wu et al. (2008b)	1999	-40%	-52%	
Zeng et al. (2008)	2000	Increase		Only worldwide emission inventory provided, however, the A2 scenario generally has ozone-precursor emissions increasing in OECD countries.

Note that the four studies cited in the TSD assumed either no change or an increase in ozone-precursor between the base-year and 2050. Figure 7 shows the extent to which this approach exaggerates future emissions of ozone precursors. The two curves with filled markers are EPA's estimate of actual U.S. NOx and VOC emissions from 1970-2011 (as already discussed in Figure 6). The dotted horizontal lines extend the 1996 base-year emissions level (used in Bell et al. (2007)) out to 2050. The solid horizontal lines extend NOx and VOC emissions for 2011 out to 2050.

The graph shows that studies that use past ozone-precursor emissions to "predict" ozone in 2050 are guaranteed to drastically exaggerate both future ozone levels and the effect of warming on future ozone levels. Note, for example, that the ozone-precursor emissions assumed for 2050 by Bell et al. (2007) and Denman et al. (2007) (which cites the Bell et al. group's results) are more than twice as high as *current* ozone-precursor emissions. Thus, these studies aren't even suitable for assessing the effect of climate warming on *current* ozone levels. Hauglustaine et al. (2005) and Liao and Seinfeld (2006) are even more fantastical, because they assume *increases* in ozone precursors in the future.

Figure 7. Studies of Ozone in 2050 Use Drastically Exaggerated NOx and VOC Emission Inventories



Notes: Bell et al. (2007) and Denman et al. (2007) report ozone results for 2050 using an ozone-precursor emissions inventory for 1996. The graph shows how this drastically exaggerates likely ozone-precursor emissions in 2050. Note that NOx and VOC emissions in 2011 were already less than half of 1996 levels and dropping rapidly.

Table 2 shows that most of the studies cited in EPA's Interim Analysis likewise assume that ozone-precursor emissions in 2050 will be the same or greater than they were during 1995 to 2001. Only four research groups—Steiner et al., Tagaris et al., Tao et al., and Wu et al.—assumed ozone-precursor emissions will be lower in 2050 than they were during 1995-2001. The largest decline assumed in a national

study[12] was the 55 percent drop in NOx emissions assumed by Tagaris et al., which used 2001 as a base year. Note, however, from Figure 7 that, compared with 2001, NOx and VOC had already declined 44 percent by 2011. At current rates of decline, both NOx and VOC will be 55 percent below their 2001 levels a year or two from now. Thus, even the most "realistic" of the ozone-climate studies are not realistic at all. At best they answer the question: "What would ozone levels be a year or two from now if the climate were a few degrees warmer than it actually will be?"

Climate Warming Will Have Much Less Effect on Future Ozone Levels than EPA Claims

It is difficult to predict with certainty how much smaller the effect of climate warming on ozone would be if EPA used a realistic ozone-precursor emission inventory to model future ozone levels, because no study to date has used anything close to a realistic ozone-precursor emission inventory for 2050. Nevertheless, one thing is certain: Reducing ozone-precursor emissions down to a small fraction of late 1990s or current levels would drastically reduce ozone levels. EPA's entire strategy to attain the ozone standard is based on exactly this premise.

Even though no one has modeled ozone in 2050 using a realistic ozone-precursor emissions inventory, studies that use somewhat lower ozone-precursor emissions than 1990s levels suggest that the "climate penalty" would be much smaller and would perhaps disappear under a realistic ozone-precursor emissions scenario.

Racherla and Adams (2009) modeled ozone in 2050 assuming that NOx and VOC would drop 61 percent and 39 percent, respectively, below 1990s levels. Given their base year and their assumed emission reductions, their study effectively compares the change in ozone in the mid-1990s with ozone levels during the last few years. Their study assumes climate warming for 2050 under the A2 scenario. In effect, Racherla and Adams (2009) assessed the effects of climate warming on ozone levels during the mid-1990s and the last few years.

Figure 2 of their study, reproduced below as Figure 8, shows the results. Each of the six panels presents the results for a different region of the United States. Let's look at the panel labeled Northeast as an example. The panel has three pairs of boxplots. Each pair shows the distribution of ozone levels in based on the A2 climate scenario for the 1990s and 2050s. That is, each pair of boxplots displays modeled ozone without and with climate warming.

The two left-most boxplots represent the 1990s and 2050s climate with 1990s ozone-precursor emissions (pc_pe and fc_pe, or "present climate_present emissions" and "future climate_present emissions"). The two middle boxplots represent 1990s and 2050s climate with *increases* in ozone-precursor emissions (NOx +33%; VOC +18%) (pc_a2e and fc_a2e, or "present climate_A2 emissions" and "future climate_A2 emissions"). Finally, the two right-most box plots represent the 1990s

12 Steiner et al. (2006) modeled parts of California and assumed VOC reductions of 50% - 70% and NOx reductions of 10% - 50% below 2000 levels, depending on the region of the state.

and 2050s climates with lower ozone-precursor emissions (NOx -62%; VOC -39%) (pc_b1e and fc_b1e, or "present climate_B1 emissions" and "future climate_B1 emissions").[13] Note the following in the Northeast panel:

In the pc_pe vs. fc_pe scenarios, which represents 1990s ozone-precursor emissions, you can see a climate penalty in that the highest ozone days are almost 10 ppb higher in the warmed climate when compared with the 1990s climate. One the other hand, with the pc_b1e and fc_b1e scenarios (which effectively represent ozone-precursor emissions during the last few years) you can see two things: First, with or without warming, peak ozone levels are much lower, more than 20 ppb lower, than for the scenarios using 1990s ozone-precursor emissions. Second, the climate penalty has nearly disappeared.[14]

The results are similar for the other areas of country. With or without warming, reducing ozone precursors from 1990s levels to recent levels results in large ozone reductions in all areas of the country. As we showed earlier, this is exactly what happened in the real world too—warmer climate, lower ozone—so it's a good thing that the model comports with this reality. Furthermore, in five of six areas of country, the climate penalty is also smaller when ozone-precursor emissions are reduced. The only exception is the southwest, where the climate penalty gets larger after reducing ozone-precursor emissions, even though overall ozone levels are still lower in the warmed climate.

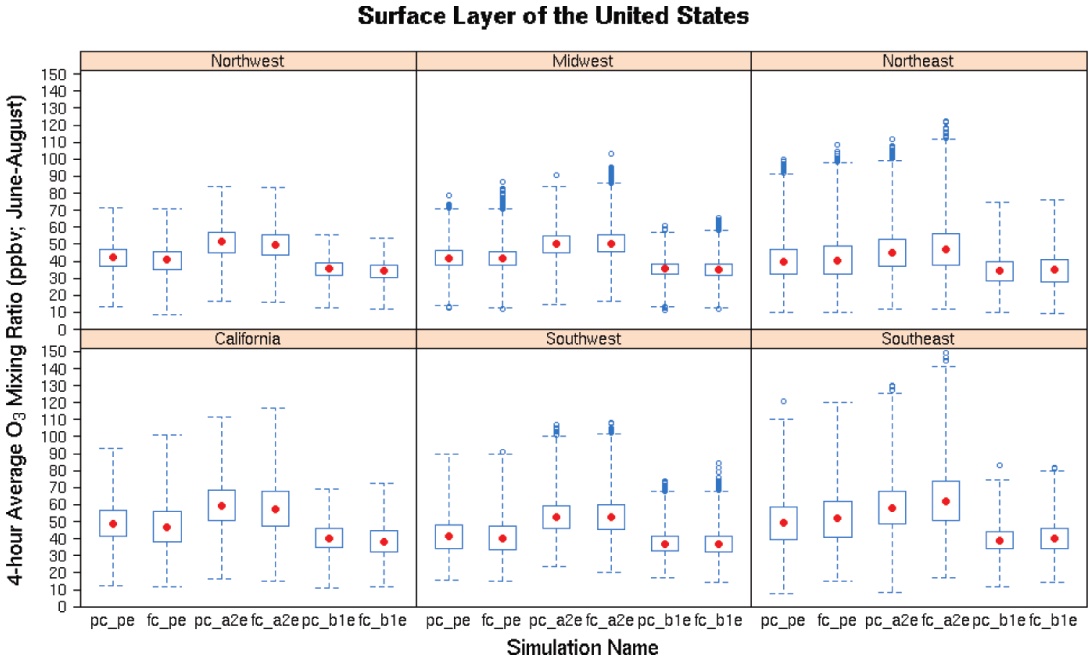
The overall lesson from Racherla and Adams (2009) is that even modest reductions in ozone-precursor emissions reduces ozone everywhere in the country, even if the climate warms substantially (as it does in the A2 scenario), and gets rid of most or all of the climate penalty in the vast majority of the country.

Wu et al. (2008) model the IPCC A1B scenario for warming in 2050 and assume that U.S. NOx and VOC emissions due to fossil fuel combustion decline by 40 percent and 57 percent, respectively, relative to 1999. Although they report a climate penalty, like Racherla and Adams (2009), they also find that reducing ozone-precursor emissions reduces the climate penalty. They conclude, "We find that a 50% reduction in U.S. NOx emissions is needed in the 2050 climate to reach the same target in the Midwest as a 40% reduction in the 2000 climate. Emission controls reduce the magnitude of this climate change penalty and can even turn it into a climate benefit in some regions."

13 Note that the "B1 emissions" applies only to ozone-precursor emissions. In all cases, the amount of greenhouse warming was based on the A2 high-warming scenario.

14 The middle pair of boxplots likewise show that if you increase ozone-precursor emissions, ozone levels rise, but we're not concerned with that here, because it is such an unrealistic scenario.

Figure 8. Racherla and Adams (2009) modeling of ozone levels with and without warming and with and without modest emissions reductions



Source: Figure 2 from Racherla and Adams (2009).

Wu et al. (2008) also find that the size of the region in which climate warming increases ozone levels shrinks as ozone-precursor emissions decrease. For example, according to Figure 9 of their paper, with 40 and 57 percent reductions in NOx and VOC (relative to 1999 emissions), the area of greatest climate change penalty (6-8 ppb higher ozone given a 2050 climate relative to a 2000 climate) shrinks by about 85 percent. On the other hand, the area where climate change has no effect or even a *benefit* (that is, *lower* ozone due to warming) greatly expands, from about one-third of the U.S. to more than half the U.S., including the entire northeast, southeast and most of the west. In particular, given these emission reductions, climate warming *lowers* ozone in most of the southeast and pacific northwest.

This is a crucial finding, because NOx and VOC emissions *have already* decreased 47 and 52 percent, respectively, relative to 1999 levels (EPA 2012a), or roughly what Wu et al. assumed for 2050. In other words, taking Wu et al.'s modeling at face value, given *current* ozone-precursor emissions, climate warming of a few degrees Fahrenheit would increase ozone in about one-third of the U.S., decrease ozone in about one-fourth of the U.S. and have no effect in the remaining five-twelfths of the U.S. But we showed earlier that ozone-precursor emissions are dropping rapidly and will be far lower in coming decades. Given the fact that Wu et al. predict a shrinking area of "climate penalty" with decreasing ozone-precursor emissions and an expanding area of "climate benefit", it seems probable that using a realistic ozone-

precursor inventory for 2050 would actually result in at worst no climate penalty and perhaps even an overall benefit of warming on ozone levels in the United States.

Finally, as noted earlier, Bloomer et al. (2009) concluded, based on observations of the real-world response of ozone to NOx reductions during the early 2000s, that a 43 percent reduction in NOx emissions from power plants reduced the climate penalty by 31 percent, from 3.2 ppb ozone/°C to 2.2 ppb ozone/°C.

Taken together, these studies lead to the conclusion that reducing ozone-precursor emissions reduces the effect of climate warming on ozone levels. These studies suggest that ozone-precursor reductions between the late 1990s and 2011 have already eliminated most of the climate penalty and perhaps even set the stage for a climate benefit in some areas. Since ozone-precursor emissions are dropping rapidly, whatever climate penalty remains will likely disappear within a decade or two, as most remaining ozone-precursor emissions are eliminated by already-adopted measures.

Contrary to EPA's conclusion in its endangerment finding, the weight of the evidence suggests that recent and future ozone-precursor reductions will soon eliminate any risk that climate warming will increase ozone levels.

As we showed earlier, ozone-precursor emissions are dropping rapidly. Even a decade from now, ozone-precursor emissions will be far lower than they are today. By 2050 the vast majority of remaining ozone precursors will have been eliminated. Because they use drastically exaggerated ozone-precursor emissions for 2050, studies of climate and ozone done to date drastically exaggerate future ozone levels and the effect of warming on future ozone levels.

In its Endangerment Finding TSD, EPA summarizes what it believes to be the weight of the evidence from ozone-climate research:

- "There is now consistent evidence from models and observations that 21st century climate change will worsen summertime surface ozone in polluted regions of North America compared to a future with no climate change (Jacob and Winner, 2009)." (TSD, p. 89)
- "Studies reviewed in the IA and Jacob and Winner (2009) indicate the largest increases in ozone concentrations due to climate change occur during peak pollution events. The locations of peak ozone episodes tend to be large metropolitan areas such as Los Angeles, Houston, and the Northeast corridor, suggesting higher increases of potentially dangerous levels of ozone over significant population centers." (TSD, p. 92)
- "Mickley et al. (2004) find that climate change projected to occur under the A1B (mid-range) scenario results in significant changes that occur at the high end of the pollutant concentration distribution (episodes) in the Midwest and Northeast between 2000 and 2050 given constant levels of criteria pollutant emissions." (TSD, p. 92)

- "Climate change is projected to increase surface layer ozone concentrations in both urban and polluted rural environments due to decomposition of PAN at higher temperatures (Sillman and Samson, 1995; Liao and Seinfeld, 2006)" (TSD, p. 90).
- "For A2 scenario in the 2050s, Bell et al. (2007) report that the projected effects of climate change on ozone in 50 eastern U.S. cities increased the number of summer days exceeding the 8-hour EPA standard by 68%. On average across the 50 cities, the summertime daily 8-hour maximum increased 4.4 ppb." (TSD, p. 92).
- "Using the A2 (high-end) emissions scenario, Hogrefe et al. (2004) find that while regional climate change in the eastern United States causes the summer average daily maximum 8-hour ozone concentrations to increase by 2.7, 4.2, and 5.0 ppb in 2020s, 2050s, and 2080s (compared to 1990s), respectively, regional climate changes causes the fourth-highest summertime daily maximum 8-hour ozone concentrations to increase by 5.0, 6.4, and 8.2 ppb for the 2020s, 2050s, and 2080s, respectively (compared to 1990s) (Hogrefe et al., 2004)." (TSD, p. 92)
- "Climate change is projected to increase surface layer ozone concentrations in both urban and polluted rural environments due to decomposition of PAN at higher temperatures (Sillman and Samson, 1995; Liao and Seinfeld, 2006)." (TSD, p. 90).

The Interim Analysis contains similar conclusions. For example:

- The Harvard group also found that peak O_3 pollution episodes are far more affected by climate change than mean values, with effects exceeding 10 ppb in the Midwest and Northeast. (IA, p. 3-3).
- "In five years of simulated summertime O_3 under both present-day and future climate conditions (with constant anthropogenic precursor pollutants), the Washington State group found future O_3 increases in certain regions, most notably in the Northeast and Southwest, with smaller increases or slight decreases in other regions (Avise et al., 2009). These climate change effects were most pronounced when considering the extreme high end of the O_3 concentration distribution." (IA, p. 3-10)
- "…the frequency of extreme O_3 events increases in the simulated future climate: over the eastern half of the United States, where the largest simulated future O_3 changes occurred, the greatest increases were at the high end of the O_3 distribution, and there was increase episode frequency that was statistically significant with respect to interannual variability (Racherla and Adams, 2008)." (IA, p. 3-5)

The results EPA cites in the TSD are based on ozone-precursor emissions that are the same as or greater than emissions during the mid-to-late 1990s. In effect, these studies asked "What would ozone levels have been back in the 1990s if the climate had been a few degrees warmer?" EPA has made it appear as if they are asking a question about future ozone levels when in fact they are merely asking a

counterfactual question about past ozone levels. Even the IA's conclusions are based mainly on studies that used 1990s ozone-precursor emissions. Where they used lower emissions, the emissions were at about *current* levels, rather than the much lower levels that will obtain in 2050.

Even in cases where a study made some attempt to reduce ozone precursors below late 1990s levels, EPA excludes these results from the IA. EPA states: "Most of the groups whose results make up this synthesis of the impacts of climate change on O_3 have also carried out additional, in most cases highly preliminary, simulations designed to investigate, to first-order, the effects of changes in climate relative to changes in worldwide and/or U.S. anthropogenic emissions of precursor pollutants. The results from these simulations are not included in the synthesis below to maintain the focus on first exploring climate change impacts alone." (IA, p. 3-14).

EPA's approach of using past ozone-precursor emissions to "predict" the effect of warming on ozone in 2050 is bizarre. No one would suggest that you should predict future climate by holding CO_2 emissions constant at current levels in your model. Likewise, the only sensible way to predict the effect of climate warming on ozone levels in 2050 is to use a realistic prediction of ozone-precursor emissions in 2050 and then run the model with and without warming. One response might be that ozone-precursor emissions in 2050 are too uncertain. Of course there are uncertainties, but the uncertainties in future ozone-precursor emissions are minute compared to the uncertainties in future GHG emissions or the effect of anthropogenic GHG emissions on climate.

As shown in Section 1, measures needed to eliminate nearly all remaining ozone-precursor emissions have already been adopted or implemented. EPA is planning still more measures to eliminate the last smidgeons of ozone-precursor emissions that might remain. Furthermore, the Clean Air Act requires attainment of the ozone standard without regard to cost and EPA has continued to tighten the ozone standard. Thus, EPA and states will adopt additional ozone-precursor reduction measures in the future.

Taken together, these considerations mean that future ozone-precursor emissions will be somewhere between a-small-fraction-of-today's-levels and zero. Those are incredibly tight error bounds when compared with the panoply of uncertainties inherent in climate prediction.

If this doesn't convince you that EPA's approach to predicting future ozone levels is backwards, try this analogy: imagine I want to understand the effect of ozone-precursor reductions on ozone levels in 2050. I expect that between now and 2050, both ozone-precursor emissions and the climate will change. So how do I isolate the effect of ozone-precursor reductions alone? Clearly I need to hold the climate constant and apply current and reduced ozone-precursor emission inventories to the same climate. But which climate should I hold constant, the climate of the late 1990s or the predicted climate of 2050? Clearly I would want the predicted climate of 2050, because I want my results to apply to the future, not the past.

Likewise, EPA and scientists want to understand the effect of climate warming on ozone levels in 2050. Could it be more obvious that they should use a realistic ozone-precursor emissions inventory for 2050 and run their models with and without the warming predicted for 2050? Instead, they used ozone-precursor emissions for 1996 to 2001 (depending on the study). In effect, the question they asked was: "How much higher would ozone levels have been back in the late 1990s if the climate had been a few degrees warmer?" This question has no policy relevance even today, when ozone-precursor emissions are about half their late 1990s levels, and is of even less relevance for the 2050s, when ozone-precursor emissions will be no more than about one-tenth of late 1990s levels.

EPA claims in its Endangerment Finding and supporting documentation "There is now consistent evidence from models and observations that 21st century climate change will worsen summertime surface ozone in polluted regions of North America compared to a future with no climate change" (TSD, p. 89). This claim is unsupportable, because it is based on studies using ozone-precursor emissions twice as high as current levels and ten times higher than likely levels in 2050. At most, EPA has shown that ozone levels would have been a bit higher in the past if the climate had been a bit warmer.

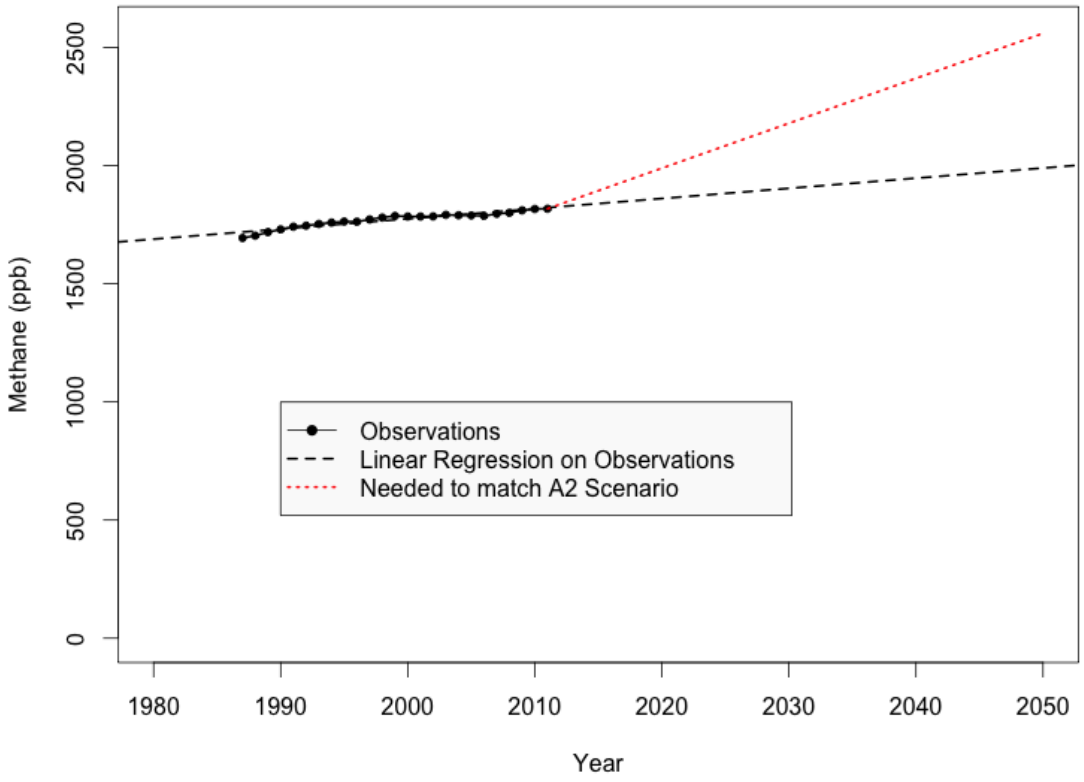
As an example of EPA's unsupportable conclusions, in its Endangerment Finding TSD, EPA states, "For A2 scenario in the 2050s, Bell et al. (2007) report that the projected effects of climate change on ozone in 50 eastern U.S. cities increased the number of summer days exceeding the 8-hour [85 ppb] EPA standard by 68%." This statement clearly leads readers to believe that climate warming is likely to cause a 68 percent increase in the number of days exceeding the 8-hour ozone standard. But even taking the model results at face value, this is only true if ozone-precursor emissions in 2050 are the same as ozone-precursor emissions in 1996. At realistic ozone-precursor emissions for 2050, their would likely be little or no increase in the number of exceedance days, because both absolute ozone levels and the increase in ozone levels due to warming would both be much lower. Once again, EPA's decision to use past ozone-precursor emissions for its modeling of ozone levels in 2050 rendered the whole exercise unsuitable for its intended purpose.

To be able to say anything about the effect of warming on ozone levels in the 2050s, EPA must run air quality models with a predicted climate for 2050 and a realistic ozone-precursor emission inventory for 2050. This is crucial, because ozone-precursor emissions in 2050 will be no more than about one-tenth 1990s levels. Indeed, more than half the emission reductions needed to go from late 1990s ozone-precursor emissions to one-tenth of late 1990s ozone-precursor emissions *have already been achieved* and the rate of emissions decline has been speeding up in recent years. Research discussed in sub-section 2.2 above shows that these ozone-precursor reductions have already eliminated most of the climate penalty and suggest that upcoming ozone-precursor reductions will eliminate what little climate penalty remains.

We close this sub-section with a brief discussion of the warming scenarios used in models of future ozone levels. Other commenters critique EPA's conclusions

regarding the extent of future warming. However, it is important to note here that most of the modeling studies on the effect of climate warming on ozone use the IPCC A2 scenario, which is among the warmest of the IPCC scenarios. The A2 scenario assumes atmospheric methane will reach 2562 ppb by 2050 (Nakicenovic and Swart 2000). Figure 9 compares the actual trend in atmospheric methane with the trend that would be necessary for methane to reach the A2 prediction by 2050. As the graph shows, a large increase in the rate of methane growth would be necessary to match the A2 scenario. We raise this issue simply to note that if anthropogenic greenhouse gases do not increase as much as the modeling assumes they will, then, even if EPA is correct about the effect of warming on ozone levels, there will not be as much warming, and hence not as much ozone, as EPA claims.

Figure 9. Actual Trend in Atmospheric Methane Compared with Trend Needed to Match A2 Scenario by 2050



Source: Methane measurements, Dlugokencky, E.J., P.M. Lang, and K.A. Masarie (2012), Atmospheric Methane Dry Air Mole Fractions from quasi-continuous measurements at Barrow, Alaska and Mauna Loa, Hawaii, 1986-2011, Version: 2012-04-05, ftp://ftp.cmdl.noaa.gov/ccg/ch4/in-situ/. A2 scenario assumptions, Nakicenovic and Swart (2000).

Note: The A2 scenario assumes methane would be 1865 ppb in 2011, compared with an actual value of 1818 ppb. Matching the A1FI methane trend (2668 ppb in 2050) would require an even more rapid increase in atmospheric methane.

3 EPA creates a false appearance that ozone must rise before regulatory agencies can take steps to reduce ozone precursors

We showed above that ozone declined substantially during the last few decades, despite 2°F of climate warming over the same period. Furthermore, we showed that ozone will decline during the next few decades, with or without warming, and that recent reductions in ozone precursor have already eliminated most of the climate penalty. Nevertheless, in its endangerment finding, EPA creates a false impression that ozone is likely to rise in the future.

For example, EPA implies that the Clean Air Act requires EPA and states to allow ozone to increase to dangerous levels before they can take action to reduce ozone, and that this means warming has to cause harm before EPA can do anything about it. In its Endangerment Finding, EPA states:

"It is important to note that controls to meet the NAAQS are typically put in place only after air quality concentrations exceeding the standard are detected. Furthermore, implementation of controls to reduce ambient concentrations of pollutants occurs over an extended time period, ranging from three years to more than twenty years depending on the pollutant and the seriousness of the nonattainment problem. Thus, while the CAA provides mechanisms for addressing adverse health effects and the underlying air quality exacerbation over time, it will not prevent the adverse impacts in the interim." (Federal Register, December 15, 2009, p. 66530)

In other words, EPA claims ozone has to rise to dangerous levels in a region in order for EPA and the region in question to gain the authority to do anything about ozone-precursor emissions. Thus, EPA argues, the fact that the Clean Air Act requires attainment is no help in preventing harm from ozone increases due to climate warming, because regulators can't do anything about the ozone increases until after the harm has already occurred.

While this may, in principle, be the legal structure of the Clean Air Act, it has little to do with the reality of how measures to reduce ozone precursors have been and are implemented. First, back in the 1970s, nearly all populated areas of the country violated the one-hour ozone standard. For example, from 1975 to 1980, between 70 and 80 percent of the nation's ozone monitoring sites violated the 1-hour ozone standard (Schwartz and Hayward 2008, 30). As a result, nearly all activity to reduce ozone after that time has been proactive, rather than responsive.

Second, EPA's continued tightening of the ozone standard has added new pressure to reduce ozone-precursor emissions. When EPA tightened the ozone standard in 1997, it set off a flurry of additional proactive actions to reduce ozone. For example, in December 2002, 33 states voluntarily submitted "Early Action Compacts" pledging to take additional steps to reduce ozone levels beyond those nominally required by EPA. As EPA noted, among the communities that joined in the EAC program were "15 communities which met the [85 ppb, 8-hour ozone] standard and want to be proactive about reducing air pollution" (EPA undated).

Third, a number of communities around the country that used to be in non-attainment of either the 1-hour, 125 ppb or the 8-hour, 85 ppb standards came into attainment at some point in the past. But coming into attainment doesn't mean sitting around and waiting for ozone to rise again. Once an area comes into attainment, it must submit a "maintenance plan" to EPA demonstrating how the area will keep ozone from rising and remain in attainment.[15] For example, measures in Kansas City's "Second Ten-Year Plan" for ozone maintenance include the following (EPA 2002):

Stationary Source Regulations: This plan relies upon continued implementation of regulations that reduce emissions from stationary sources and include the following rules:

- 10 CSR 10-2.040 Maximum Allowable Emission of Particulate Matter from Fuel Burning Equipment Used for Indirect Heating
- 10 CSR 10-2.080 Emission of Visible Air Contaminants from Internal Combustion Engines; rescinded March 18, 2003 (68 FR 12827). See 10 CSR 10-6.220
- 10 CSR 10-2.090 Incinerators
- 10 CSR 10-2.100 Open Burning Restrictions
- 10 CSR 10-2.150 Time Schdule for Compliance
- 10 CSR 10-2.205 Control of Emissions from Aerospace Manufacture and Rework Facilities
- 10 CSR 10-2.210 Control of Emissions form Solvent Metal Cleaning
- 10 CSR 10-2.215 Control of Emissions from Solvent Cleanup Operations
- 10 CSR 10-2.220 Liquefied Cutback Asphalt Paving Restricted
- 10 CSR 10-2.230 Control of Emissions from Industrial Surface Coating Operations
- 10 CSR 10-2.260 Control of Petroleum Liquid Storage, Loading, and Transfer
- 10 CSR 10-2.290 Control of Emissions from Rotogravure and Flexographic Printing Facilities
- 10 CSR 10-2.300 Control of Emissions from the Manufacturing of Paints, Varnishes, Lacquers, Enamels and Other Allied Surface Coating Products
- 10 CSR 10-2.310 Control of Emissions from the Application of Underbody Deadeners
- 10 CSR 10-2.320 Control of Emissions from the Production of Pesticides and Herbicides
- 10 CSR 10-2.340 Control of Emissions from Lithographic Printing Facilities
- 10 CSR 10-2.360 Control of Emissions from Bakery Ovens
- 10 CSR 10-6.075 Maximum Achievable Control Technology Regulations
- 10 CSR 10-6.220 Restriction of Emission of Visible Air Contaminants

15 You can find ozone maintenance plans for dozens of communities all around the U.S. by Googling "ozone 'maintenance plan'".

Fourth, and most important, nearly all ozone-precursor emissions come from nationally regulated sources—mobile sources such as cars, trucks, construction and farm equipment, trains, and planes, stationary sources such as power plants, refineries, and chemical plants, and area sources, such as paints, coatings and solvents. Measures to reduce ozone-precursor emissions from these sources are national in scope and improve ozone in all areas, regardless of whether their ozone levels are high, medium, or low, and regardless of their ozone attainment status. As noted above in sections 1 and 2, ozone-precursor emissions have been dropping rapidly on a national basis and existing requirements will eliminate nearly all remaining ozone-precursors during the next couple of decades.

A few Google searches with terms such as "ozone attainment plan", "ozone maintenance" or "ozone state implementation plan" will demonstrate the enormous amount of activity and resources that have been in the past and are currently being brought to bear by all levels of government to reduce ozone-precursor emissions and attain the federal ozone standard.

EPA's implication that there are areas of country where ozone will have to rise before anything can be done about it couldn't be further from the truth. EPA has been pre-emptively reducing ozone-precursor emissions for decades in all areas of the U.S. and will continue to do so. Local and state governments are taking additional actions to reduce the few sources of ozone precursors that are not under federal control. Thus, there has not been, is not now, nor will there be in the future any location in the country that is just sitting around waiting to go into non-attainment.

4 Ozone at Current, Historically Low Levels Is Not A Significant Human Health Concern, and Future Ozone Levels Will Be Far Lower, Regardless of Climate Warming

We showed above that without climate warming ozone will decline substantially in the future, and that with climate warming ozone will still decline substantially, but slightly less than without warming.

Here we show that regardless of the effect of warming on ozone levels, ozone at current, historically low levels is already low enough not to be a health concern. There will be even less reason for concern about ozone at the much lower levels that will obtain in the future. Thus, regardless of the effect of climate change on ozone levels, there is no reason for concern over the effects climate warming on ozone-related health effects.

In the TSD for its Endangerment finding, EPA cites its most recent *Air Quality Criteria Document for Ozone and Related Photochemical Oxidants* (Environmental Protection Agency 2006) as the source for its conclusions regarding the effects of ozone on health. Below we critique the claims in that document and also discuss

more recent work since 2006.[16] The topics we discuss can be summarized as follows:

Observational studies generate false indications of risk where no risk in fact exists. EPA's claims for ozone's most serious health effects—premature death and respiratory and cardiovascular hospitalizations—are based on the results of observational epidemiology studies. We show that observational studies generate false indications of risk through data dredging and publication bias. This happens because (1) observational studies inherently allow great flexibility in modeling choices with little or no means of external validation, (2) large datasets are always filled with small chance correlations and the putative effects of ozone are within the same range as these chance correlations, and (4) from among the thousands or millions of superficially plausible models of air pollution and health, researchers seek out models that give statistically significant results. As a result, observational studies tend to confirm the preconceptions of the researchers, rather than provide realistic information on health effects. In cases where observational studies have been tested against randomized controlled trials, the observational study results are nearly always falsified. In its criteria documents and associated reports on ozone's 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.

The claim that ozone can kill at real-world exposure levels is biologically implausible. Laboratory studies with hundreds of individual animals representing several different species show that animals do not die, even when exposed for the equivalent of many years to ozone at levels nearly ten times greater than the highest ambient levels. These results make it biologically implausible that ozone at real-world outdoor levels could be causing premature death in humans.

Selective characterization of evidence. In its characterization of ozone's health effects, EPA selectively emphasizes studies and portions of studies reporting harmful ozone effects, while downplaying studies reporting no effects or apparently protective ozone effects. This creates an impression that the evidence for harm from ozone at real-world levels is far more robust and consistent than the full weight of the evidence suggests.

Studies of low-level ozone exposure with human volunteers demonstrate that an 8-hour standard of 85 ppb is more than stringent enough to protect human health with an adequate margin of safety, even from the most mild health effects EPA claims for ozone. A few studies with human volunteers have assessed the effects of ozone at levels below 80 ppb. These studies find small, temporary reductions in lung function that EPA classifies as adverse. But EPA is mistaken on

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16 We submitted comments to EPA on the Ozone Criteria Document and hereby incorporate those comments by reference. See J. Schwartz, "Comments on EPA's Proposal to Adopt A More Stringent Ozone Standard," comments submitted to Docket EPA-HQ-OAR-2005-0172, October 9, 2007, available at http://www.aei.org/files/2007/10/09/20071101_SchwartzOzoneComments.pdf.

two accounts. First, the subjects in the study had to exercise for the equivalent of six consecutive gym workouts in less than 7 hours just to elicit small, temporary, and medically insignificant effects on lung function. Outside these artificial laboratory conditions, no one is active long enough or intensely enough to elicit even these tiny effects.

Second, EPA ignores the difference between personal exposure and ambient-monitor levels when interpreting these low-exposure studies. As a result of this difference, a 60 ppb personal ozone exposure in a laboratory study is equivalent to at least 100 ppb measured at an ambient ozone monitor. After correcting for this bias, even if EPA is correct that the minor effects seen in laboratory studies are adverse, these effects do not occur until ozone levels are well above a level equivalent to 85 ppb as measured at an ambient monitor.

The overall result of the above considerations is that the 85 ppb 8-hour ozone standard protects human health with plenty of room to spare. Peak annual ozone levels are already below this level in about 90 percent of the country. Continued ozone-precursor reductions will ensure that the entire country will be well below this level by the time any significant additional warming occurs. Thus, even if warming causes ozone to decrease a bit less than it otherwise would have, ozone levels around the U.S. will still be below a level of concern for human health, with or without climate warming.

Observational studies create false indications of risk where no risk in fact exists. All of the evidence for harm from ozone at realistic levels comes from observational studies and can therefore be discounted.

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 associated Staff Paper (EPA 2007a) 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 an 85 ppb, 8-hour standard. 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. Unlike controlled clinical or laboratory studies, which can 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 that are statistical figments, rather than real effects.

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 dredging 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 dredging refers to the risk that observational studies can become statistical fishing expeditions that turn up chance correlations, rather than real causal relationships. Think of the statistical models that researchers use to control for bias in observational studies as having lots of "dials" or "knobs" that 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.[17] 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. (Anderson et al. 2004)

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

Estimation of very weak associations in the presence of measurement error and strong confounding is inherently challenging. In this situation, prudent epidemiologists should recognize that residual bias can dominate their results. Because the possible mechanisms of action and their latencies are uncertain, the biologically correct models are unknown. *This model selection problem is exacerbated by the common practice of screening multiple analyses and then selectively reporting only a few important results*. (emphasis added) (Lumley and Sheppard 2003)

17 Publication bias is a well-documented problem in a range of disciplines. See, for example, Montori et al. (2000) and Thornton and Lee (2000).

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. (Anderson et al. 2004)

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.[18] In a recent review of observational studies, Young and Karr reported that 12 recent randomized trials tested 52 different claims from observational studies (Young and Karr 2011). All 52 claims were contradicted by the randomized controlled trials.

A number of epidemiologists and statisticians 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 (Taubes 1995; Ioannidis 2005; G. D. Smith 2001). 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 (Anderson et al. 2004; M. L. Bell, Dominici, and Samet 2005; Ito 2003; Keatinge and Donaldson 2006; Koop and Tole 2004).

Because 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* (Ioannidis 2005). The 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.

An additional reason to discount epidemiologic studies is their lack of biologic plausibility. Laboratory studies with animals suggest that ozone is not deadly, even at concentrations many times greater than ever occur in ambient air. Researchers have exposed monkeys, rats, and other species to very high levels of ozone (as high

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18 For example, hormone replacement therapy and Vitamin A turned out not to reduce risk of cardiovascular disease, following a low-fat diet turned out not to reduce risk of heart disease or colorectal and breast cancer, and calcium supplements didn't reduce the risk of osteoporosis (Beresford et al. 2006; Howard et al. 2006; Kolata 2006; Moolgavkar 2005; Prentice et al. 2006; G. D. Smith 2001; Taubes 1995).

as 1000 ppb) for the equivalent of years, yet none of the animals have died.[19] At a 2005 meeting of EPA's Clean Air Science Advisory Committee to discuss a draft of EPA Ozone Criteria Document, Charles Plopper, a professor at UC Davis, expressed skepticism regarding the claim that ozone causes premature mortality, stating "I'm trying to look at it as a biologist and trying to figure out whether [ozone] exposure kills people. And I've never killed a rat in 35 years…and never killed a monkey in 35 years. And I've been accused of using too high [ozone] levels…So I'm trying to figure out does this even make any sense from a biology point of view?"[20]

EPA's Selective Characterization of evidence

Although EPA's Ozone Criteria Document (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 (Environmental Protection Agency 2006, 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, the NMMAPS study (M. L. Bell et al. 2004) 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 (see Figure 3 in Bell et al. (2004)). A more recent reanalysis of the NNMAPS data has only amplified these concerns (R. L. Smith, Xu, and Switzer 2009).

Nevertheless, EPA 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 removed from the analysis. Examination of Figure 3 in Bell et al. (2004) suggests that one extreme outlier city and two or three more moderate outliers are driving the statistical association of ozone and mortality as well. The CD

19 There are dozens of such studies. A few include, (C. Y. Chen et al. 2003; Barr et al. 1990; Barr et al. 1988; Chow, Plopper, and Dungworth 1979; Dodge et al. 1994; Harkema, Plopper, and Hyde 1987; M. G. Lee et al. 2008; Moffatt et al. 1987; Wilson, Plopper, and Dungworth 1984)

20 CASAC meeting transcript, December 8, 2005, 148.

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.

In its recent Integrated Science Assessment for ozone EPA continues to ignore evidence against ozone having a causal role in mortality associations. EPA asserts that the mortality effects for ozone have been found at concentrations well below the current 75 ppb standard, citing an analysis of the NMMAPS data that excludes high-ozone days from its analysis (EPA 2012b; Michelle L. Bell, Peng, and Dominici 2006). However, in a follow-up study, Bell et al. (2007) restricted the analysis to days with low ozone, the variability of ozone effects across communities actually widened. When the analysis was restricted to days with ozone less than 20 ppb, the range of individual city ozone-mortality associations for a 10 ppb increase in ozone ranged from -20 percent to +30 percent. It is not plausible that such low ozone exposures could be causing large increases in mortality in some cities and large decreases in mortality in others. With such large variations and such biologically implausible results, the most plausible interpretation is that these are not real ozone effects, but statistical artifacts.

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 EPA-commissioned meta-analyses (M. L. Bell, Dominici, and Samet 2005; Ito, De Leon, and Lippmann 2005; Levy, Chemerynski, and Sarnat 2005) 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" (CD, 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 more likely due 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." (Goodman 2005)

The CD's cursory treatment of publication bias also points up the selective way in which the CD marshals 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 evidence that publication bias inflates 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, that ozone is unlikely to be associated with daily mortality (Koop and Tole 2004). 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 Paper.

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" (Health Effects Institute 2003). Likewise, Ito (2003), in the same report, concluded:

"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."

Likewise, Smith et al. (2001) similarly warn:

"From a statistical point of view, the common epidemiological practice of choosing variables (including lagged variables, co-pollutants, etc.) that maximize the resulting effect estimates is a dangerous approach to model selection, particularly when the effect estimates are close to 0 (i.e., RR close to 1.0)."

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. More recent work suggests that failing to account for model selection bias can make air pollution effects appear statistically significant when in fact they are not (Roberts and Martin 2010).

Keatinge and Donaldson (2005) showed that changes in adjustment for weather can cause the apparent effect of ozone on short-term mortality to disappear. When they

allowed in their model for cumulative effects of heat stress over several days, as well as 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.

The CD's density plots indirectly show that consideration of publication and model-selection bias would have greatly reduced 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 Health Effects Institute's recent APHENA study only amplifies concerns regarding EPA's exaggeration of ozone-mortality associations (Katsouyanni and Samet 2009). In single-pollutant models of ozone and mortality, only 12 of 24 models resulted in a statistically significant relationship between ozone and death in the U.S. Furthermore, when modeling risk of death in those over and under 75 years of age, 10 of 24 models gave statistically significant results for those under 75, and 6 of 24 for those over 75. In models that also controlled for particulate matter, ozone was no longer statistically significant in any of the models. None of these results suggests robust or consistent evidence for ozone as a cause of premature mortality.

Overall, EPA's conclusion of robustness and consistency of ozone associations with mortality and other health effects is mistaken, and its presentation of the evidence creates an appearance of consistency and robustness that does not exist in the full range of research results.

The paragraphs above discuss EPA's general problem of mischaracterizing evidence. Below we point out some additional cases in which EPA's ozone Criteria Document 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 (McConnell et al. 2002). 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 Paper makes a similar claim (p. 3-24). 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.[21]

The CD notes that asthma risk was 3.3 times greater for children in high-ozone communities playing three 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 (in order to match the 30 percent lower risk of asthma observed for the full cohort).

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)—at the time had by far the highest ozone levels in the country.[22] The study was based on ozone levels during 1994-97, when these areas violated the old 1-hour ozone standard dozens of times per year and violated the 8-hour, 85 ppb ozone standard more than a hundred 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 that even the old 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 EPA should have drawn: First, overall, higher ozone levels reduce the risk of developing asthma.[23] Second, the federal 1-hour, 125 ppb and 8-hour, 85 ppb ozone standards protect against the development of asthma with a huge margin of safety, even in the most physically active children. The CD and Staff Paper should not have created the impression that a more stringent ozone standard would reduce children's risk of developing asthma.

Another important result from the Children's Health Study is that even after a exposure from birth to ozone exceeding the 1-hour 125 ppb standard more than a hundred days per year, ozone had no effect on teenagers' lung development or lung function (Gauderman et al. 2004).

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21 Based on 1-hour ozone levels and this result was statistically significant. 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, i.e., a hair short of statistical significance.

22 They still have the highest ozone levels in the country, but ozone levels there have dropped substantially since the CHS years and are now closer to ozone levels elsewhere.

23 We do not advocate this view. We merely point out that if the study has uncovered a causal relationship between high ozone and increased asthma for children playing three or more team sports, then there is no basis for not considering as causal the statistically significant relationship between high ozone and lower asthma in less-active children.

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)" (CD, p. 8-44) shows that ozone is associated with increased medication use. 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 covariate. 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" (CD, 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.[24]

CARB/Kaiser Central Valley Study. This time-series study reported a statistically significant *decrease* in acute health effects with higher ozone levels (van den Eeden et al. 2002). The CD does not mention this study.

Laboratory studies with human volunteers indicate that an 8-hour ozone standard of 85 ppb is protective of human health with room to spare

In both the CD and more recent Integrated Science Assessment (ISA) (EPA 2012b), EPA asserts that laboratory studies with human volunteers provide direct evidence that ozone causes adverse effects at levels below the 85 ppb, 8-hour ozone standard, and even the newer 75 ppb ozone standard. In the CD EPA places special emphasis on studies by Adams (2006; 2002) because at the time they were the only studies that examined respiratory effects associated with ozone exposures for several hours at levels below 80 ppb. The ISA also cites more recent studies that also assessed effects of low ozone levels on human volunteers (Kim et al. 2011; Schelegle et al. 2009).

In these studies, healthy young-adult college students were exposed to ozone at various concentrations (60, 70, 80, and 87 ppb, depending on the study) for 6.6 hours while exercising, and their lung function and subjective symptoms were measured several times during the exposure period. Group-average changes in lung function with 60 ppb ozone were very small. After 6.6 hours, FEV_1 (forced expiratory volume in one second) declined by about 1.7 to 2.7 percent (declines were not statistically significant), depending on the study, returning back to normal within 1 hour after ozone exposure ended. Subjective symptoms were not affected by exposure to ozone.

Schlegle et al. (2009) assessed a 70 ppb exposure and reported a mean reduction in FEV_1 of about 5 percent that took a few hours to return back to normal. Total Symptom Score also increased, from a maximum of about 4 with ozone-free air to a maximum of about 13 with 70 ppb ozone. This is on a scale from zero to 160, so the change in symptoms was quite small.

These changes are small and clinically insignificant. However, EPA expressed concern over the fact that a few subjects experienced larger temporary reductions in

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

lung function. Regarding the Adams studies, EPA reported that after 6.6 hours, 2 of 30 subjects in the Adams studies experienced temporary lung-function reductions in FEV_1 of 10 percent or more, when exposed to ozone at 60 ppb (EPA 2007b, 37828).[25] More recently, Schlegle et al. (2009) also reported that 6 of the 31 subjects in that study likewise experienced temporary FEV_1 reductions greater than 10 percent at both 60 and 70 ppb. Based on these results, EPA concludes that standards of 85 ppb or 75 ppb do not sufficiently protect people from ozone. In its ISA, EPA concludes: "Though group mean decrements are biologically small and generally do not attain statistical significance, a considerable fraction of exposed individuals experience clinically meaningful decrements in lung function" (ISA p. 6-18).

This conclusion is unwarranted for two reasons. First, because of the well known difference between ozone concentrations measured ambient monitoring stations and actual personal exposures to ozone while outdoors, the personal ozone exposures in the Adams, Schlegle et al., and Kim et al. studies were equivalent to ambient-monitor ozone levels of 100 ppb or more. After accounting for the personal exposure-to-ambient monitor ratio, it is clear that ozone does not have adverse effects at ambient-monitor concentrations below 85 ppb.

Second, even without accounting for the personal exposure-to-ambient monitor ratio, EPA had to make a heroic stretch to find adverse effects in the laboratory ozone studies. As discussed in more detail below, the studies used unrealistically extreme amounts of exercise (equivalent to four or five gym workouts in a row) and the few cases with FEV_1 declines of greater than 10 percent could easily have been due to within-subject variability, rather than ozone.

i. Ignoring the difference between personal exposure and ambient-monitor ozone levels causes EPA to great exaggerate ozone's health effects. Once this difference is accounted for, there is no evidence for adverse effects of ozone at levels below 85 ppb (as measured at ambient monitors).

A great deal of evidence indicates that personal ozone exposures—the amount of ozone in the air people actually breathe into their lungs—even while outdoors, are much lower than ambient ozone levels measured at ambient ozone monitors. The reason is that ambient ozone compliance monitors are generally placed several feet above human head-height and away from surfaces, in order to avoid interferences from people and surfaces near the ground that could affect the fidelity and consistency of the ozone measurements. Ozone is very reactive and any nearby surfaces (such as clothing or the ground) reduces the amount of ozone in the air that people actually breathe into their lungs.

Evidence comparing ambient ozone concentrations with personal exposures includes the following:

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25 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.

- Trained technicians in eastern Los Angeles County wore personal 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 (Johnson et al. 1996). Personal exposures while outdoors averaged 41 percent lower than hourly ambient ozone levels reported at the nearest monitors. The ratio of personal to ambient ozone level was the same whether the technician was near or away from roadways.
- Outdoor workers in Mexico City experienced average personal ozone exposures 60 percent lower than ambient-monitor levels in a study of thirty-nine shoe-cleaners (O'Neill et al. 2003). All ozone exposures in this study took place outdoors.
- In a study of thirty-six children in Tennessee, those in the top 25 percent for time-spent-outdoors nevertheless experienced personal ozone exposures 80 percent lower than levels measured at ambient monitors (K. Lee et al. 2004).
- A study in Baltimore had a trained technician perform scripted activities to simulate a typical daily schedule of a senior citizen while carrying a personal ozone monitor (Chang et al. 2000). The study reported that personal ozone exposure during the summer averaged 33 percent lower than ambient-monitor levels while outdoors near a roadway, and 11 percent lower while outdoors away from roadways.
- A companion study to the one above measured personal ozone exposures of 15 senior citizens during summer in Baltimore for a total of 12 days each (Sarnat, Koutrakis, and Suh 2000). The highest personal exposures—reflecting more time spent outdoors—were well below ambient-monitor levels. While the highest ambient-monitor level on any day was 54 ppb the highest personal ozone exposure for any of the 15 people in the study was 21 ppb (60 percent lower) and the second highest was 17 ppb (68 percent lower).[26] As the authors noted, "[P]ersonal exposures to O_3 [ozone], NO_2, and SO_2 were extremely low. Seventy percent of the measured personal O_3, NO_2, and SO_2 values were below their respective LOD [limit of detection], even when ambient concentrations were well above their LOD" (Sarnat, Koutrakis, and Suh 2000, 1188).
- Liu et al. (1997) found that a group of forty children and adults in Alpine, California, experienced average personal ozone exposures 75 percent lower than ambient levels.

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26 In this study, both personal and ambient ozone levels were reported as 24-hour averages. To get a rough idea of equivalent 8-hour and 1-hour levels, multiply by 1.5 and 2.0, respectively. Thus, a 24-hour average of 54 ppb corresponds roughly to an 8-hour level of 85 ppb, and a 1-hour level of 11 ppb. In other words, peak ambient ozone levels during the study were about as high as the old 8-hour standard and well above the new 75 ppb 8-hour standard.

- 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 averaged 61 and 58 percent below the respective ambient levels in the two areas (Geyh et al. 2000).
- A study of 158 asthmatic children in Mexico City reported that personal ozone exposures averaged 77 percent less than ambient-monitor concentrations (Ramirez-Aguilar et al. 2008). The highest personal ozone exposure—reflecting the most time spent outdoors—for any child was 52 percent lower than the *highest* ambient-monitor ozone level and 7 percent lower than the *median* ambient-monitor ozone level during the study.

This difference between ambient ozone concentrations and personal exposures is key for interpreting the laboratory studies. The ozone levels used in laboratory studies such as Adams, Schlegle et al., and Kim et al., are *personal exposures*. To compare them to equivalent ambient-monitor levels, one must account for the fact that personal ozone exposures while outdoors are typically at least 40 percent lower than ozone levels measured at ambient monitors. Or, to turn this around, ozone levels measured at ambient monitors are typically at least 1.67 times greater than concomitant personal outdoor exposures.

Table 3, below, translates the personal ozone exposures used in the laboratory studies into equivalent concentrations at ambient compliance monitors. Because ambient-monitor levels are at least 1.67 times personal exposures, Table 3 uses a conversion factor of 1.67 to go from the personal exposure levels in the Laboratory studies to equivalent ambient levels. The table gives both the average and peak level for each exposure pattern.

Table 3. Comparison of personal ozone exposures used in the Adams, Schlegle et al. and Kim et al. studies with equivalent ambient-monitor concentrations

Laboratory personal ozone exposure protocols			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	0	0	0
Triangular	40	50	66	84
Square	60	60	**100**	100
Triangular	60	90	**100**	*150*
Triangular	70	90	**117**	*150*
Square	80	80	**134**	*134*
Triangular	80	150	**134**	*250*

Notes: All values are in parts per billion (ppb). Bold entries signify ozone exposures that exceeded the 8-hour, 75 ppb or 85 ppb ambient standards. Italicized entries signify exposures that also exceeded even the old 1-hour, 125 ppb standard. A "square" exposure pattern means that subjects breathed a constant ozone concentration for the 6.6-hour experiment. 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 comparing apples to oranges when it assumes the effects of, say, 60 ppb ozone in the laboratory provides information on the health effects of 60 ppb ozone measured at an ambient compliance monitor. In fact, 60 ppb in the laboratory is equivalent to 100 ppb as measured at an ambient monitor. After converting the personal exposures in Adams (2006) to equivalent ambient levels, we can see that the effective ozone exposures in Adams (2006) were actually much greater than EPA assumes. For example, to get a personal exposure of 60 ppb, the ambient concentration would need to be at least 100 ppb. If anything, Adams (2006) and more recent similar studies (Kim et al. 2011, Schlegle et al. 2009) show that even ozone levels substantially greater than the 8-hour, 75 or 85 ppb standards have little or no effect on people's lung function.

EPA is aware of the difference between ozone concentrations measured at ambient monitors and actual personal exposure concentrations, but ignores this difference in interpreting laboratory studies of the effects of low ozone levels. In its Ozone Criteria Document (EPA 2006) EPA devotes pp. 3-56 to 3-76 to a discussion of personal exposure vs. ambient concentration and cites most of the articles we cite above in the bullet points. However, EPA cites these papers to support the (correct) claim that personal exposures and ambient-monitor levels are *correlated*, meaning that personal exposures tend to rise and fall in concert with ambient-monitor levels. EPA notes this correlation to support the contention that ambient monitors provide a valid measure of ozone exposure for the purposes of observational epidemiology studies. However, EPA omits the fact that these same studies also show that personal exposures, even while outdoors, are *much lower* than ozone levels measured at ambient monitors.

There is, however, one instance where EPA explicitly compares outdoor personal exposures to outdoor ambient-monitor levels. In this case, EPA notes that Brauer and Brook (1997) reported that the personal ozone exposure of farm workers in the lower Fraser Valley (Canada), who spent all of their time outdoors, was only 4 percent lower than ambient monitor levels (Environmental Protection Agency 2006, 3–74), implying that personal exposure it not much lower than ambient-monitor levels.

EPA's characterization is misleading in three ways. First, Brauer and Brook themselves point out that their personal ozone measurements have a large error and likely a positive bias as well.[27] Second, ambient ozone levels were extremely low

27 "For some individuals in Group 3 [farm hands working outdoors] personal/fixed ozone ratios were near zero, while for others the ratios were above two (Fig. 1). These extreme values can be attributed in part to random measurement error which

in this study—about 15 to 30 ppb on most days and never greater than 50 ppb (as measured at ambient monitors). Third, difference between personal exposure and ambient-monitor ozone was greatest on days with the highest ozone. For example, the five highest-ozone days ranged from 39 to 50 ppb (all other days ranged from 8 to 34 ppb) personal exposure averaged 22 percent lower than ambient-monitor levels (compared with 4 percent lower overall).[28]

In summary EPA mentions the difference between ambient and personal ozone levels when doing so helps EPA make a case for the validity of the observational epidemiology studies (i.e., when EPA notes the *correlation* between personal and ambient ozone), but generally ignores the personal vs. ambient difference when doing so would vitiate EPA's case for harm from low-level ozone (i.e., EPA ignores the fact the outdoor personal ozone exposures are much lower than ambient-monitor levels when interpreting the results of the Adams laboratory studies). Furthermore, in the one case where EPA does compare personal to ambient ozone levels, EPA cites only a single outlier study (in which the study authors themselves cast doubt on the validity of the measurements) to create a false impression that there is little difference between personal and ambient ozone levels.

ii. Even without accounting for the personal exposure-to-ambient monitor ozone ratio, the laboratory results suggest that ozone does not have adverse effects below 85 ppb

EPA glosses over is how difficult it is to induce even the small ozone effects observed in the laboratory studies, despite the fact that the ozone levels in these studies were effectively much greater than the original 8-hour ozone standard and even the old 1-hour standard.

It is true, as EPA notes, that a couple of subjects experienced FEV_1 declines greater than 10 percent at the 60 ppb exposure in the Adams studies. Figures 10 and 11 display individual results for, respectively, men and women, at the 60 ppb triangular exposure. As the graphs show, one man experienced an FEV_1 decline of 12 percent after 6.6 hours, and one woman experienced a decline of 21%.

Figure 10. Adams (2006) data for men

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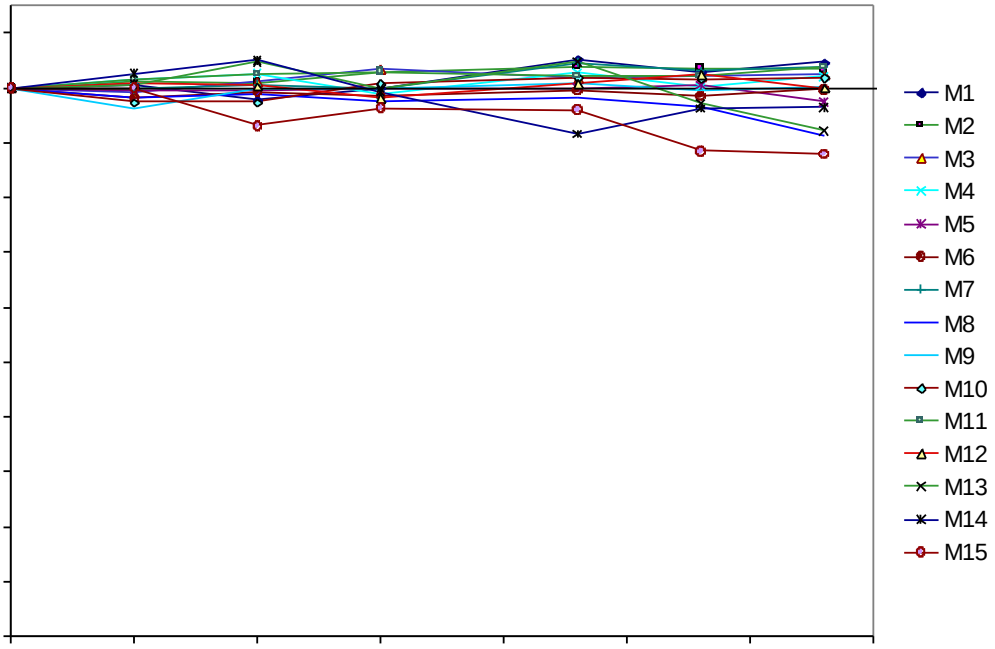
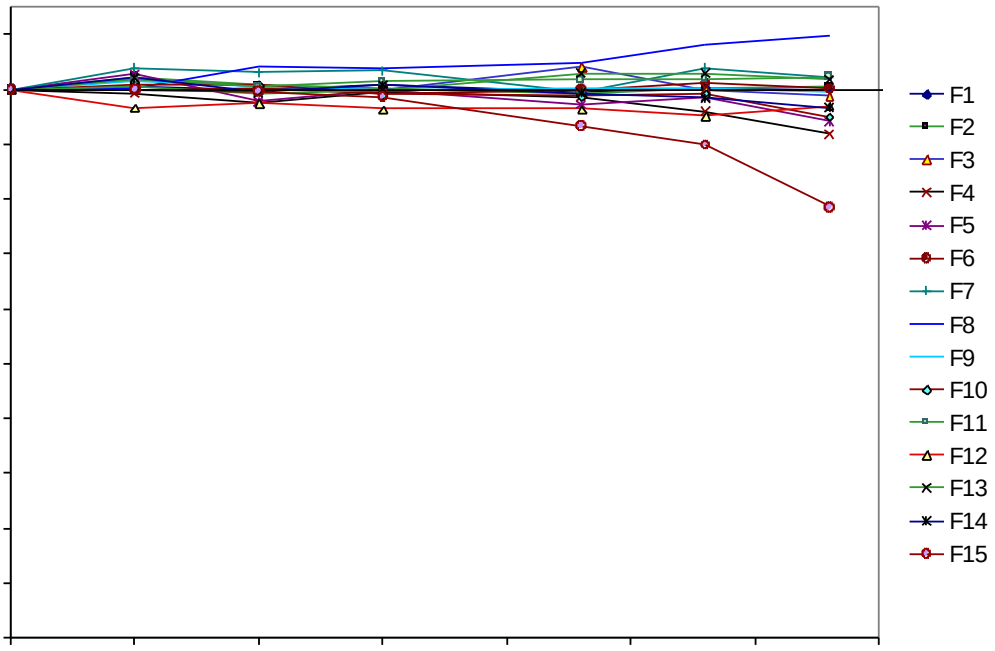
we have estimated at 35% (Brauer and Brook 1995)…The high personal-fixed ratios are more difficult to explain and possibly indicate local variability in ozone concentrations or the effect of HNO_3 or some other unidentified interference affecting personal ozone samplers." Brauer and Brook (1997), 2116-17.

28 See Figure 2(a) in Brauer and Brook (1997).



Source: Data provided by William Adams, Professor Emeritus, UC Davis.

Figure 11. Adams (2006) data for women



Source: Data provided by William Adams, Professor Emeritus, UC Davis.

First, note once again that the ozone exposure was equivalent to at least 100 ppm when translated into ambient-monitor terms, and was therefore effectively at least 18 percent greater than an 8-hour ozone standard of 85 ppb. Thus, even these effects required a relatively high ozone exposure. It certainly does not provide evidence of any effects at ozone levels below an ambient monitor level of 85 ppb.

Second, note that it took more than five hours of continuous ozone exposure before even the most sensitive people began to show any FEV_1 reduction. The subjects spent 50 minutes of every hour performing relatively vigorous aerobic exercise on a treadmill or stationary bicycle. Each 50-minute bout was roughly equivalent to a gym workout. The full 6.6-hour exercise regimen was similar to going on an all-day hike or going to the gym six times in a row in one day.[29] This level of exercise was

29 The average young adult at rest has a respiration rate on the order of 12-18 breaths per minute and a heart rate of about 60-80 beats per minute. In the Adams studies the participants spent 5 hours out of the 6.6 hours of the study (50 minutes per hour, with a 35 minute lunch break in the middle) exercising so as to keep their respiration rate at an average of 26-27 breaths per minute and their heart rate at 123-125 beats per minute. For people in their early twenties (as were the subjects in the Adams studies), the target heart rate for aerobic exercise is 117-165

necessary to raise people's respiration rate high enough so that they would breathe in enough ozone to affect their FEV_1. In other words, it took an unrealistically lengthy bout of relatively vigorous exercise even to achieve the small effects that were observed at the 60 ppb personal exposure. To see how unrealistic, the breathing rates in the laboratory studies can be compared with real-world breathing rate data for the general population from Brochu (2006).

In the three laboratory studies, the subjects inhaled air at a rate of 20 liters per minute per square meter of body surface area. This translates into inhalation rates of about 55 cubic meters or air per day (m^3/day) for men and 46 m^3/day for women. In the Brochu (2006) data, the 99th percentile of inhalation rate for any age group is 24 m^3/day for men and 19 m^3/day for women (both for ages 18 to 30).

Since ozone dose is proportional to air inhalation rate, this demonstrates that the ozone doses used in the Adams, Schlegle et al., and Kim et al. studies are much higher than real-world humans would ever experience. And even so, these studies were able to elicit minimal effects on lung function and symptoms.

There is an additional reason why this issue of lengthy exercise is important. The Adams study was performed with physically fit exercise physiology majors from UC Davis. The Schlegle et al. and Kim et al. studies were performed with similar physically fit young adults. EPA claims that these people, being healthy, are less likely to experience significant effects of ozone and that children and the elderly would be more sensitive to ozone's effects. As a result, EPA claims, these laboratory studies place only a lower limit on the effects of low ozone exposures. But this claim ignores the fact that children, the elderly, and those with respiratory diseases are *not capable* of exercising vigorously for the hours in a row necessary to elicit even the small ozone effects reported by Adams. Indeed, as the Brochu (2006) data show, for children and the elderly, 99th percentile inhalation rates are much lower than for the 18 to 30 age group: 12 m^3/day for children ages 3 to 10, and 18 m^3/day for adults over 60. Thus, contrary to EPA's claim, the results of Adams, Schlegle et al., and Kim et al. apply even less to children and the elderly than to young adults.

Conclusion

EPA's ozone modeling is based on 1990s ozone-precursor emissions, making it invalid for assessing the effects of climate warming on ozone levels in the future, when ozone-precursor emissions will be far lower. Indeed, even *current* ozone-precursor emissions are only about half the levels used in EPA's studies.

Research based on more realistic levels of future ozone-precursor emissions shows that ozone levels will be much lower in the future, regardless of warming, and that the effect of warming on ozone—the "climate penalty"—has already been greatly reduced. Since ozone-precursor emissions are dropping rapidly, whatever climate

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beats/minute (60%-85% of the recommended maximum safe heart rate for one's age).

penalty remains will likely disappear within a decade or two, as most remaining ozone-precursor emissions are eliminated by already-adopted measures.

Taken together the weight of the evidence indicates that EPA has exaggerated future ozone levels, the effects of warming on ozone levels, and the health risks from any given level of ozone. In reality, future ozone levels will be below a level of concern for human health, regardless of whether the climate warms.

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Appendix G

All-Cause and Cause-Specific Mortality by Socioeconomic Status Among Employed Persons in 27 US States, 1984–1997

Kyle Steenland, PhD, Sherry Hu, MS, and James Walker, PhD

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Abstract

Several large cohort studies in the United States have shown that mortality rates are higher among those of low versus high socioeconomic status (SES). Higher levels of standard risk factors for those with low SES do not appear to entirely account for this fact.1–5

Several studies have also considered temporal changes in mortality by SES and have shown that cardiovascular mortality has been decreasing faster for higher-SES groups from the 1950s to the 1980s.6–10 Few data on SES and mortality have been published in recent years. The only study to have yielded more recent data was based on the American Cancer Society population, which has a higher SES than the general population. We examined whether previously observed SES differences in mortality persisted during the 1990s and how these differences changed over time in a population reasonably representative of the US population: employed persons aged 35–64 years in 27 states during the period 1984–1997.

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