COMPETITIVE ENTERPRISE INSTITUTE

The Clear Skies Initiative is Hazy

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Executive Summary

Proponents of the Clear Skies Initiative claim that it will avert thousands of deaths annually by reducing emissions from coal-fired power plants. Environmental activist groups counter that Clear Skies will kill tens of thousands annually because it does not go far enough to curb emissions. This paper counsels skepticism on the root epidemiological premise behind the claims of both proponents and opponents of Clear Skies—the assumption that fine particulate ($PM_{2.5}$) pollution kills people at any level of exposure.

The paper makes four main points. First, it is unlikely that $PM_{2.5}$ at current levels is causing any mortality. EPA based its $PM_{2.5}$ standard largely on the American Cancer Society (Pope et al.) study. The odd variations that study found in the association between $PM_{2.5}$ and mortality defies biological explanation. For example, Pope et al. found no association between $PM_{2.5}$ levels and mortality for women, for persons with more than a high school education, and for persons between the ages of 60 and 69.

Second, Clear Skies (like its more aggressive alternative, Sen. James Jeffords' (I-VT) Clean Power Act) assumes without evidence that there is no threshold below which $PM_{2.5}$ poses no health risks. This unsupported assumption leads to greatly exaggerated estimates of the mortality effects of current $PM_{2.5}$ levels.

Third, if $PM_{2.5}$ were the dire threat that Clear Skies advocates assume, then Clear Skies would do little to combat it. Clear Skies does not target resources at areas with the highest $PM_{2.5}$ levels. Rather, it seeks to save lives by achieving small incremental reductions in $PM_{2.5}$ levels everywhere. This makes about as much sense as establishing a national anti-obesity program designed to help every overweight person lose one pound rather than to help the most dangerously obese individuals lose tens of pounds.

Fourth, when the Clear Skies benefits model is run retrospectively, i.e., backward from 2000 to 1980, it fails to show any benefits corresponding to the known decreased trend in $PM_{2.5}$ exposures. Significant reductions in $PM_{2.5}$ over the past two decades did not reduce cardiopulmonary mortality risk at all.

I. Introduction

The Bush Administration's proposed Clear Skies Initiative (CSI) has been touted as the panacea for mitigating a myriad of air pollution-related environmental impacts in one fell swoop. Supposedly, CSI will save thousands of lives, greatly reduce lung-related health effects, make fish safe to eat, save the Chesapeake Bay, clean up the Great Smoky Mountains, and perform other wonders. However, none of the claimed benefits can be substantiated when scrutinized under a rudimentary scientific microscope.

This paper examines the science behind the CSI health benefit claims—the same science that, ironically, underlies environmental advocacy groups' condemnation of CSI for not doing enough to control power plant emissions.

It is axiomatic that environmental policy should be based on science. Does CSI meet this test? The results of this analysis will show that the proposed sulfur dioxide emissions reductions will not save 12,000 lives in 2020, as claimed by the Bush Administration, and that the estimate could be as low as 400 *even if one accepts the use of the controversial American Cancer Society (Pope et al.) study upon which the mortality risk estimates are based*. By the same token, this analysis shows that CSI will not kill tens of thousands of people as a result of its being less draconian than Sen. James Jeffords' (I-VT) "Clean Power Act" (S. 366), which many environmental groups support.

CSI targets a single air pollution source category—the coal utility industry[1]—rather than areas with the highest pollution levels. For example, the top six counties for PM pollution are all in California, which has no coal-fired power plants.

II. Weak Science Behind EPA's $PM_{2.5}$ Standard

Over the past 30 years, the utility industry has greatly reduced emissions of sulfur dioxide (SO_2), one of the three primary emissions of concern from power plants. The others were coarse particulate matter (PM_{10}) and nitrogen oxides (NO_X). There are few, if any, U.S. urban areas that are not in compliance with the National Ambient Air Quality Standards (NAAQS) for SO_2 based on monitored data. In fact, ambient SO_2 levels are far below the NAAQS, as shown in Figure 1.

Both CSI and the Clean Power Act would require additional reductions in SO_2 emissions. SO_2 is a precursor of fine particulate ($PM_{2.5}$) pollution, and hence a contributor to the annual average $PM_{2.5}$ levels in areas of the country that exceed 15 micrograms per cubic meter (15 $\mu g/m^3$)—the standard EPA set in 1997. Both CSI and Clean Power Act advocates claim that current $PM_{2.5}$ levels pose a major health threat. However, the science underpinning such claims and EPA's standard is weak.

EPA based its $PM_{2.5}$ standard largely on the study by Pope et al. That study claimed to discover a significant correlation between long-term $PM_{2.5}$ levels and mortality.[2] In July 2000, the Health Effects Institute (HEI), an independent research organization jointly sponsored by government and industry, published a reanalysis of Pope et al.[3] Former EPA Administrator Carol Browner stated that HEI "re-evaluated the science and

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[1] Those who expect "regulatory certainty" from CSI are not looking at it in historical context. Only 25 years ago, the Carter Administration focused on making our vast coal reserves the cornerstone of an energy policy for achieving less dependence on imported oil. Agencies undertook in-depth environmental and societal impact analyses to evaluate the efficacy of this policy initiative. President Carter appointed a special committee, headed up by Dr. David Rall, to address global warming, acid rain, urban air pollution, and mine safety issues among others. The only thing "certain" is that, if enacted, Clear Skies will overturn what remains of the U.S. Government's previous commitments to coal-based power.

[2] C.A. Pope et al., "Particulate Air Pollution as a Predictor of Mortality in a Prospective Study of U.S. Adults," *American Journal of Respiratory Critical Care Medicine*, vol. 151, pp. 669-674 (1995).

[3] D. Krewski et al., "Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality" (Cambridge, Massachusetts: Health Effects Institute, 2000).

confirmed our results."[4] More recently, Pope et al. together with some participants in the HEI reanalysis published a study that reported that a 10 $\mu g/m^3$ increase in long-term $PM_{2.5}$ levels was associated with a 4 percent increase in risk of cardiopulmonary death.[5] However, several features of these studies suggest that other "co-factors" (confounding variables such as lifestyle, income, and other pollutants) were responsible for the apparent association between $PM_{2.5}$ and mortality.[6] For example, there was no significant association between $PM_{2.5}$ levels and mortality for persons with more than a high school education, for women, and for persons between ages of 60 and 69. Similarly, $PM_{2.5}$ was associated with mortality risk for former smokers, but not current smokers or people who never smoked, and for moderately active people but not either sedentary or very active people. These odd correlations between $PM_{2.5}$ and mortality defy biological explanation.

In addition, when the reanalysis combined all cofactors that might influence mortality risks, the association between $PM_{2.5}$ and mortality was not significant. When the reanalysis included sulfur dioxide levels as a potential co-factor, the $PM_{2.5}$ mortality link disappeared.

III. The CSI Mortality Model and the No Threshold Assumption

In short, both CSI and the Clean Power Act assume, without solid evidence, that $PM_{2.5}$ *at current levels* kills. Even more dubiously, they assume there is *no threshold* for mortality effects from exposure to fine particles. In other words, both proposals suppose that fine particles kill, no matter how low ambient air concentrations get. They assume that the only safe level is zero.

The case for Clear Skies rests on an interconnected sequence of assumptions:

1. $PM_{2.5}$ pollution kills tens of thousands of people annually.
2. Reductions in SO_2 emissions translate directly into lower concentrations of sulfate fine particles.
3. Lower levels of sulfate particles translate directly into lower levels of $PM_{2.5}$.
4. The small predicted incremental $PM_{2.5}$ reductions (generally less than 1 $\mu g/m^3$ across the nation) translate directly into reduced air pollution-related health effects, regardless of existing levels of exposure.

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[4] Carol Browner, National Press Club, October 3, 2000, www.epa.gov/opa/admspchs.
[5] C.A. Pope et al., "Lung Cancer, Cardiopulmonary Mortality, and Long-tern Exposure to Fine Particulate Air Pollution," *Journal of the American Medical Association*, vol. 287, pp 1,132- 1,140 (2002).
[6] For further discussion, see Kay Jones and Ben Lieberman, *The Ongoing Clean-Air Debate: The Science Behind EPA's Rule on Soot*, Competitive Enterprise Institute, June 2001, pp. 5-6; Joel Schwartz, *Particulate Air Pollution: Weighing the Risks*, Competitive Enterprise Institute, April 2003, pp. 16-17.

Obviously, at some level of exposure, any inhaled substance will impair respiratory function and health. However, it does not necessarily follow that *any* level of exposure impairs health. More often than not, it is "the dose that makes the poison."[7]

Epidemiological studies such as Pope et al. cannot be used to establish an effects threshold because of the wide scatter in the data, as shown in Figure 2. This scatter is typical of epidemiological studies and is due to the natural variation of health status, behavior, and environmental exposures among individuals in any population. Well-controlled clinical studies are the only way to determine such thresholds, but one cannot administer controlled doses of air pollution to thousands of human subjects over multi-year periods. In any event, the fact that Pope et al. data do not reveal a threshold is not evidence that a threshold does not exist.

Contrary to EPA's no-threshold assumption in the CSI benefits analysis, EPA, in the course of its deliberations on the $PM_{2.5}$ standard, actually tried to identify a threshold in the data, as shown in Figure 2.

In the staff paper published prior to promulgation of the proposed standard, EPA concluded that there was a threshold at a mean (annual average) of 15 $\mu g/m^3$. EPA's standard is based on this exposure limit. However, EPA failed to note an error in the Pope paper, which mistook for a mean $PM_{2.5}$ concentration what was actually the city-by-city median concentration. Hence, the statistical equivalent of an annual median of 15 $\mu g/m^3$ is an annual mean of approximately 18.7 $\mu g/m^3$. The difference is due to the fact that the day-to-day $PM_{2.5}$ measurements, examined over a year, have a right-skewed, rather than a normal or bell-shaped, distribution. The mean of such observations is always higher than the median. The 18.7 $\mu g/m^3$ annual average concentration should have been EPA's default threshold, that is, its proposed ambient standard. In fact, this author has pointed this out to EPA.

EPA admitted the error but did not modify its proposal from a 15 $\mu g/m^3$ median concentration to what should have been an annual mean equivalent of 18.7 $\mu g/m^3$. EPA has not been able to justify the 15 $\mu g/m^3$ standard since the error was revealed.

EPA stood behind the guidance of the Science Advisory Board (SAB) concerning threshold assumptions when considering cost benefit analyses of alternative ambient standard options, i.e., ambient standard options above and below 15ug/m^3. EPA's Technical Addendum on Clear Skies claims that, "the SAB subsequently advised EPA that there is currently no scientific basis for selecting a threshold of 15ug/m^3 or any other specific threshold for PM-related health effects."[8] However, absence of a scientific basis for selecting one threshold rather than another is not positive evidence—given the

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[7] For a technical discussion, see R.L. Smith et al., "Threshold Dependence of Mortality Effects for Fine and Course Particles in Phoenix, Arizona," *Journal of the Air and Waste Management Association*, vol. 50, no 8 (2000), pp. 1367-79; and S.H. Moolgavkar and E.G. Luebeck, "A Critical Review of the Evidence on Particulate Air Pollution and Mortality," *Epidemiology*, vol. 7, no. 4 (1995), pp. 420-8.

[8] EPA, *Technical Addendum: Methodologies for the Benefit Analysis of the Clear Skies Initiative*, September 2002, p. 24.

limitations of epidemiological studies—that no threshold exists. It would appear that EPA took this more narrow SAB guidance out of context to support the zero threshold assumption, which underpins the CSI analysis.

It is not possible to precisely reproduce the CSI benefits results or conduct alternative analyses, because the overall model is not available for inspection or use by the public. However, some simplifying assumptions yield results comparable to the CSI projections. It is disconcerting that the CSI benefits model did not provide results on a Standard Metropolitan Statistical Area (SMSA) basis (Clear Skies Act, 2002). Had this been the case, city-by-city benefits could have been computed. Instead, the CSI air quality benefits projections were based upon some 5,000 grid cells covering the nation. Hence, in order to analyze the policy implications for St. Louis, for example, one has to first find the coordinates of the grid cells that overlay St. Louis and then review four separate files, each containing over 5,000 entries, to determine the baseline and the projected 2010 $PM_{2.5}$ air quality levels. These files became the basis for the benefits maps featured in the CSI proposal. EPA should have been much more transparent in presenting its CSI modeling results to the public.

Rather than conduct a reanalysis at the census tract level, this paper uses SMSA population data. It estimates incremental air quality benefits based on the average $PM_{2.5}$ reductions for a cross section of six cities—Cleveland, Pittsburgh, Knoxville, Birmingham, St. Louis, and Chicago—representing the regions of the U.S. affected by the CSI pollution reduction strategy. The range of the CSI benefits alone in 2020 among these cities is 1.1 to 2.9 $\mu g/m^3$ with an average of 1.8 $\mu g/m^3$. The range of the baseline CAA benefit without CSI is 0.5 to 2.5 $\mu g/m^3$ with an average of 1.3 $\mu g/m^3$.[9] The baseline CAA benefit is that associated with the acid rain emissions reduction provision of the Act.

This paper also assumes that the baseline mortality rate for the population over age 30 is the same everywhere, rather than county specific, as in the CSI model. This rate, based on 1998 vital statistics data, is 1,412 deaths per 100,000 persons over 30 (NCHS 2002). The mortality estimates at the SMSA level simply become the product of the SMSA population times the CSI $PM_{2.5}$ reduction increment times the mortality risk per microgram per cubic meter of $PM_{2.5}$. The unit risk based on the CSI documentation is 6.5 deaths per 1 $\mu g/m^3$ per population of 100,000. As will be shown, the simplified approach yielded almost identical results to CSI where parallel data were available.

EPA uses the zero threshold assumption throughout the CSI analysis, as shown in Table 1 and Figure 3, which come directly from the CSI documentation. When the exposed populations are summed, the totals are 295.5 million in 2010 and 320.7 million in 2020, or the entire nation's projected population. The data in Table 1 can be used to crosscheck the CSI benefits analysis by incorporating the CSI mortality benefits model. The model

[9] This is consistent with Abt Associates Inc.'s estimate of the change in annual mean $PM_{2.5}$ levels after a 75 percent reduction in power plant emissions. See Abt Associates Inc., *Particulate-Related Health Benefits of Reducing Power Plant Emissions*, October 2000, p. 3-4, http://abtassociates.com/reports/particulate-related.pdf

calculates the incremental number of lives saved per incremental change in $PM_{2.5}$ over and above the baseline annual death rate for the population over age 30, that is, the Pope et al. age baseline. The CSI modeling exercise was done at the census track level from an exposed population perspective, but had to rely on county level 1995 baseline mortality levels. When adjusted for age, the baseline mortality rate does not vary much across the nation. The midpoint concentration within the tabulated range in Table 1 represents the change in exposure for the population exposed within the increment given in the Table. The results of this simple macro analysis yielded 6,135 and 11,771 lives saved in 2010 and 2020 respectively. The CSI rounded results claim 6,000 lives saved in 2010 and 12,000 lives saved in 2020. Hence, the macro analytical approach is assumed to be a good approximation of the detailed CSI model output.

IV. Alternative Mortality Forecasts

Figure 3 also verifies the zero threshold assumption in that the benefits decrease rapidly as the threshold assumption is increased incrementally from zero upwards. For example, the graph shows that if the $PM_{2.5}$ threshold were set at the annual standard of 15 ug/m^3, the CSI 2020 benefit drops by 80 percent, from 11,700 to 2,200 lives saved per year.

In order to put the CSI mortality benefits claim in a broader perspective, we need to establish a baseline mortality rate under EPA's zero $PM_{2.5}$ threshold assumption. This paper uses EPA's air quality and population data for the 309 U.S. SMSAs (EPA 2000) for computational purposes. It uses U.S. Census data to partition the population data for persons over the age of 30, who comprise 58 percent of the total population. Hence, the $PM_{2.5}$-related deaths in each SMSA are simply the product of its population (in 100,000s) x 0.58 x the SMSA's $PM_{2.5}$ annual concentration x the unit risk of 6.5 deaths per 1 $\mu g/m^3$ per population of 100,000. When these $PM_{2.5}$-related deaths are summed across all 309 SMSAs, we find that $PM_{2.5}$ killed 110,800 people in the year 2000! This astounding estimate would make $PM_{2.5}$ air pollution responsible for 13 percent of all deaths due to heart and lung disease. It would make $PM_{2.5}$ the fifth leading cause of death in the U.S., ahead of all accidental deaths, an inherently implausible result.

Without any further age adjustment, the 2020 projected baseline air pollution death rate would be approximately 129,500 deaths per year, using the same population growth rate assumed in the CSI benefits analysis. The CSI projected benefit of 11,700 lives saved, when compared with EPA's implicit $PM_{2.5}$ baseline mortality estimate, seems paltry—CSI only reduces the national risk by 9 percent. No wonder environmental groups, operating from the same assumptions, accuse CSI of not doing enough!

This same analysis, however, can be performed using a non-zero threshold assumption. Table 2 shows these comparative results. If the threshold is set at the current standard of 15 $\mu g/m^3$, CSI reduces the current mortality risk by 14 percent while the current Clean Air Act provision reduces it by 30 percent. If the $PM_{2.5}$ standard had been set at EPA's perceived threshold of 18.7 $\mu g/m^3$ as previously discussed, the estimated CSI and CAA mortality reductions would be 6 percent and 12 percent respectively.

CSI claims that the targeted SO_2 reductions will significantly help achieve attainment of the $PM_{2.5}$ standard. This is true for areas in the Midwest and the East that are currently only marginally above the 15 $\mu g/m^3$ standard level, for example, less than 17 $\mu g/m^3$. However, a review of EPA's publication of county-level non-attainment areas (U.S. EPA) shows that the 11 most highly impacted counties and their associated SMSAs would receive little or no benefit from CSI. These 11 counties represent all of the metro areas with $PM_{2.5}$ levels above 20 $\mu g/m^3$. The top six counties are all in California, which has no coal-fired power plants. These data and the associated excess mortality data under the CSI benefit increments are shown in Table 3. Only one of the 11 metro areas—Knoxville—would be brought into attainment by CSI. Table 3 also shows the excess mortality rate under the three threshold assumptions discussed here.

It is important to note that, when compared to the total CSI mortality estimates, these 11 metro areas are responsible for more than 50 percent of all of the estimated excess mortality due to $PM_{2.5}$ above 15 $\mu g/m^3$ and 98 percent of the excess mortality above 18.7 $\mu g/m^3$. CSI only reduces the excess mortality risk estimates above 15 $\mu g/m^3$ by 1,016 lives or 9.1 percent among these 11 most highly impacted metro areas in the nation.

V. Clearly Inefficient

Clear Skies and Clean Power target a single pollution *source category*—the coal-fired utility sector—rather than the *areas with the worst particulate pollution.* Therefore, the CSI policy will not reduce by any significant measure the exposure to fine particulates in the 11 most highly impacted cities in the nation where most of the estimated excess mortality might occur. For instance, California has some of the highest levels of particulate pollution, but no coal-fired power plants. CSI is analogous to a national diet plan whose goal is reducing every overweight person's weight by one pound. Clearly, more lives would be saved by a plan that focused on helping the most obese people reduce their weight by tens of pounds.

In addition to the unrealistic, if not implausible, mortality estimates that emerge from analyses based on a zero effects threshold for $PM_{2.5}$, a mitigation policy that aims at a small benefit for everyone in the nation is flawed from a risk management perspective. In preventive health care, resources are targeted at the higher risk segments of the population, where the association between apparent cause and effect is strongest, because that is where interventions are likely to save the most lives. The CSI strategy does not fit this simple concept. It does not target resources at those areas with the highest $PM_{2.5}$ levels (see Table 3).

To see how ineffective CSI would be, even if its zero threshold assumption were correct, it may be useful to consider an analogy. The scientific literature establishes a linear relationship between mortality and overweight or obesity. Ironically, the same American Cancer Society Cohort from which Pope et al. derived the $PM_{2.5}$ mortality function was also the basis for a detailed study of obesity and mortality (Calle et al., 1999).

According to that study, the risk of premature mortality for persons with a body mass index (BMI) of 40 is approximately 1.6 times that of people with average weight—a BMI of 24. This roughly compares the mortality risk of a 150 lb. person to that of a 240 lb. person. These indices are the averages for males and females combined. The authors state that the mortality risk increases proportionately to BMI. Hence, the mortality risk per pound of excess body weight above the average BMI can be calculated. The excess risk is 0.64 percent and 0.51 percent per additional pound for men and women, respectively. The population-weighted risk is then 0.57 percent per pound. The projected number of overweight/obese people over age 30 in 2020, based on 1999/2000 survey data, is 128,409,000 persons (NCHS 2000). The excess mortality rate is then 1412 x .00573 or 8.1 deaths/100,000 persons who are overweight and over age 30. In other words, there are 10,389 deaths per pound of excess body weight per year.

Since a 3,500-calorie reduction in food intake equates to a one-pound weight loss, the study implies that if every overweight person over age 30 would eat 3,500 calories less per year, we would reap about the same benefit as CSI, and at no public cost. In fact, if we assume the dieter spends less on food he does not eat, this one-pound weight loss plan would save lives and money at the same time. Would this plan pass muster as a national preventive-medicine strategy with the Surgeon General? Obviously not. The rational objective is to reduce the risk to those most at risk. The goal of any public health strategy should be to obtain larger weight reductions from the most seriously obese people, not a one-pound loss from all persons with a BMI above the statistical average. Yet that, in effect, is what CSI attempts to do—save lives by reducing $PM_{2.5}$ by about 1 $\mu g/m^3$ everywhere.

VI. Retrospective Analysis

The CSI mortality benefits model can be run retrospectively (i.e., backwards from 2000 to 1980) as well as prospectively (i.e., from 2000 to 2020). EPA has documented that atmospheric fine particle sulfates declined by 50 percent from 1980 to 2000 (U.S. EPA, Sept. 2002). In those regions of the nation where coal-fired utilities exist and where sulfur dioxide reductions would have the greatest effect, approximately 50 percent of $PM_{2.5}$ is sulfate. This 50 percent sulfate reduction translates into a 25 percent reduction in $PM_{2.5}$. The mean $PM_{2.5}$ exposure concentration in 2000 can be computed based on the total estimated mortality assuming a zero threshold—110,843 deaths—and the 2000 population over 30 years of age—158,977,000. This calculated national average exposure concentration is 10.7 $\mu g/m^3$. The retrospective average exposure $PM_{2.5}$ level in 1980 therefore would have been 14.3 $\mu g/m^3$ assuming a 25 percent $PM_{2.5}$ reduction from 1980 to 2000. The age-adjusted population over age 30 was estimated to be 122,400,000. The retrospective air pollution-related mortality estimate is therefore 113,771 deaths per year. These 1980 and 2000 mortality estimates can be compared to the vital statistics on leading causes of death for these two end points.

This paper uses 1985 vital statistics data because 1980 data were not available in a compatible disease category format. Table 4 shows the comparative data. The air pollution mortality estimate is a subset of the combined heart and lung disease

(cardiopulmonary-related) deaths. This is, in fact, the hypothesis espoused in Pope et al. If $PM_{2.5}$ is the cause of air pollution related mortality, then the historical reduction of $PM_{2.5}$ should have reduced the fraction of cardiopulmonary deaths associated with $PM_{2.5}$ exposure. It is clear from the fraction calculated in Table 4—13.6 percent in 1985 and 13.3 percent in 2000—that this is not the case. If the air pollution-related mortality estimate for 1980 is adjusted to reflect the $PM_{2.5}$ reduction between 1980 and 1985, the 1985 air pollution mortality would be 106,610 deaths, or 12.7 percent of the 1985 total cardiopulmonary deaths.

This analysis must of course assume that any confounding factors have not changed over the intervening years. However, the CSI projections to 2020 are predicated on this same assumption.

The bottom line result of this retrospective analysis is that a significant historical reduction in $PM_{2.5}$ did not reduce the air pollution-related cardiopulmonary mortality risk at all. One would have expected the 1985 $PM_{2.5}$-related mortality estimate to be much higher, commensurate with the higher $PM_{2.5}$ exposure at that time. This retrospective analysis undermines the credibility of the CSI model in terms of the assumed linear relationship between $PM_{2.5}$ levels and associated cardiopulmonary mortality. A valid model used to project human health risk must be able to match historical data when the model is run backwards in time.

Conclusion

Why did the Bush Administration go down the dubious path outlined in this paper? Clear Skies melds two popular but misguided concepts. First is the idea that pollution emissions must be minimized even in the absence of evidence of harm—based on the notion that further incremental emission reductions of any pollutant always translate into a calculable increment of public health benefit. Thus, both CSI and its "green" critics claim that a 1 ug/m^3 of $PM_{2.5}$ exposure will kill people throughout the nation, regardless of anyone's current level of exposure.

Second is the idea that "market-based" cap-and-trade strategies create "regulatory certainty" and deliver environmental benefits at predictable low cost.

This analysis provides several counterpoints, which question the environmental benefit claims contained in the CSI initiative. A major flaw in the CSI benefits analysis is the assumption that there is no threshold below which $PM_{2.5}$ has no health effects. When this assumption is applied faithfully, the air pollution-related mortality estimates become nonsensical. CSI's purported benefits become tiny in comparison.

EPA's claim that CSI will bring the nation into $PM_{2.5}$ attainment is highly questionable, especially when the areas with the highest exposures are examined. Of the 11 most highly impacted urban areas, only one is brought into attainment by CSI coupled with future CAA benefits.

EPA's assumption that a small environmental exposure applied to a large population constitutes a large risk is also dubious, as can be seen when we consider the parallel between the purported mortality risk of $PM_{2.5}$ exposure and the risk of being overweight. Reducing body weight by one pound for those slightly overweight will hypothetically save as many lives as the CSI initiative. Even if correct, far more lives could be saved if the most obese persons lost significantly more than one pound. Likewise, focusing pollution reductions on the areas with the most serious air pollution and choosing the least-cost reductions first would save the most people for any given level of expenditure.

Finally, when the CSI model is run retrospectively, from 2000 to 1980, it fails to show any benefits relative to the observed decrease trend in $PM_{2.5}$ exposures. This finding strongly suggests that CSI's assumption regarding the mortality effects of $PM_{2.5}$ derived from Pope et al. is invalid.

References

1. Technical Addendum: Methodologies for the Benefit Analysis of the Clear Skies Initiative, Clear Skies Act of 2002, U.S. EPA, Sept. 2002.

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3. Peak Air Quality Statistics for the Six Principal Pollutants by Metropolitan Statistical Area 2000, U.S. EPA.

4. National Vital Statistics Report, Vol. 50, No 3, Mar 2002.

5. List 1 Counties Exceeding the PM 2.5 NAAQS, 1999-2001, U.S. EPA 2002.

6. Calle Eugenia, Michael Thun, Jennifer Petrelli, Carmen Rodriguez, Clark Heath, "Body-Mass Index and Mortality in a Prospective Cohort of U.S. Adults," *New England Journal of Medicine*, Vol. 341 No 15, Oct. 1999.

7. National Health and Nutrition Examination Survey, Table 70, National Center for Health Statistics, CDC, DHHS, 2002.

8. 2001 Status and Trends Report, EPA 454/k-01-001, U.S. EPA, Sept. 2002.

Figure 1

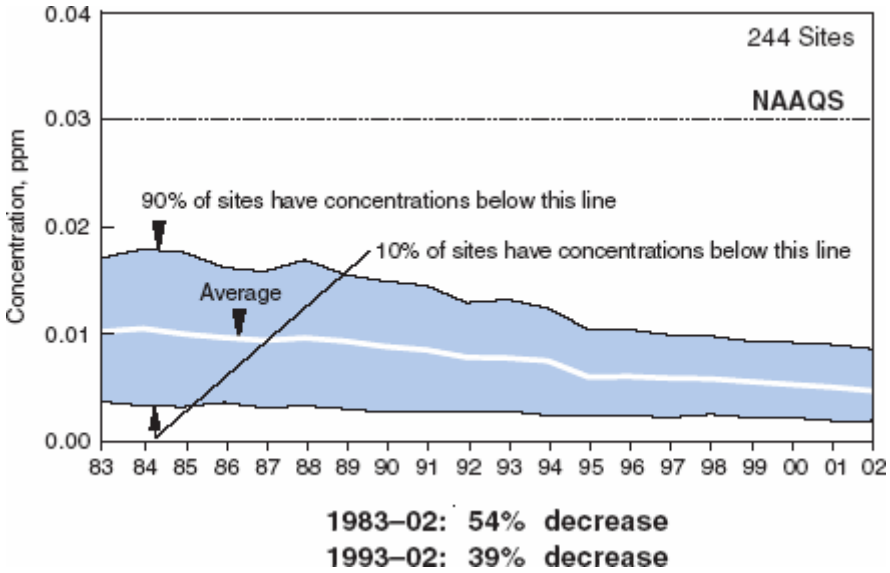

0.04
0.03
0.02
0.01
0.00
Concentration, ppm
244 Sites
NAAQS
90% of sites have concentrations below this line
10% of sites have concentrations below this line
Average
83 84 85 86 87 88 89 90 91 92 93 94 95 96 97 98 99 00 01 02
1983–02: 54% decrease
1993–02: 39% decrease

TABLE 1

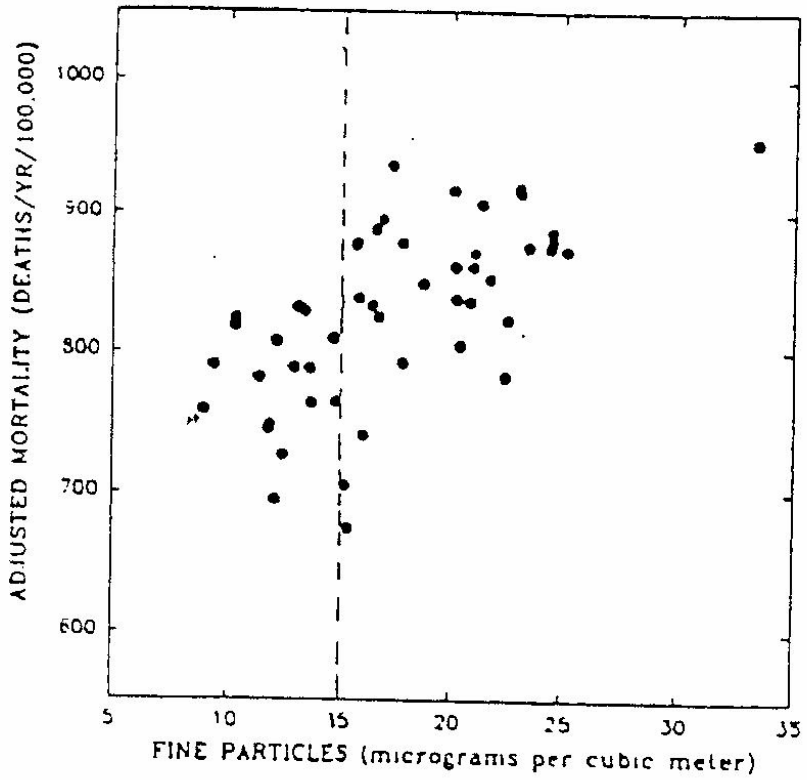
Exhibit 14
Distribution of $PM_{2.5}$ Air Quality Improvements Over 2010 and 2020 Population Due to the Clear Skies Act

Change in Annual Mean $PM_{2.5}$ Concentrations ($\mu g/m^3$)	2010 Population		2020 Population	
	Number (millions)	Percent (%)	Number (millions)	Percent (%)
$0 < \Delta PM_{2.5}$ Conc ≤ 0.25	83.4	28.2%	80.6	25.1%
$0.25 < \Delta PM_{2.5}$ Conc ≤ 0.50	51.8	17.5%	18.4	5.7%
$0.50 < \Delta PM_{2.5}$ Conc ≤ 0.75	70.2	23.8%	29.0	9.0%
$0.75 < \Delta PM_{2.5}$ Conc ≤ 1.0	49.0	16.6%	44.0	13.7%
$1.0 < \Delta PM_{2.5}$ Conc ≤ 1.25	36.5	12.4%	49.9	15.5%
$1.25 < \Delta PM_{2.5}$ Conc ≤ 1.50	3.9	1.3%	21.8	6.8%
$1.50 < \Delta PM_{2.5}$ Conc ≤ 1.75	0.7	0.2%	26.2	8.2%
$1.75 < \Delta PM_{2.5}$ Conc ≤ 2.0	-	-	26.7	8.3%
$\Delta PM_{2.5}$ Conc > 2.0	-	-	24.1	7.5%

* Totals may not sum due to rounding.

FIG 2

RELATIONSHIP BETWEEN ADJUSTED MORTALITY AND PM
THE AMERICAN CANCER SOCIETY STUDY (POPE ET AL., 1



Age-, sex-, and race-adjusted population-based mortality r
1980 plotted against median fine particulate air pollution level
to 1983. Data from metropolitan areas that correspond appro
areas used in prospective cohort analysis.

TABLE 2

PROJECTED MORTALITY RATES UNDER ALTERNATIVE THRESHOLD ASSUMPTIONS 2020 ESTIMATES

Threshold Assumption Cases	Concentration Basis ($\mu g/m^{3)}$	2020 PM 2.5 Mortality Rates (deaths/yr) Total no CAA & CSI	CAA Reduction	CSI Reduction
EPA/CSI	0	129,465	NC*	11,771
NAAQS	15	19,467	5,956	2,791
Alternative Std	18.7	6,325	751	354

*NC = Not Calculated

Table 3							
		THE CSI BENEFITS IN THE ELEVEN METRO AREAS WITH					
		THE HIGHEST PM 2.5 LEVELS					
Estimated Death Rates/Year							
	Design	2020 Adult	Based on Alternative Thresholds of			CSI PM 2.5	
	Value[1]	Population[2,3]				Reduction	Lives Saved
Metro Area	($\mu g/m^3$)	(millions)	0 $\mu g/m^3$	15$\mu g/m^3$	18.7$\mu g/m^3$	($\mu g/m^3$)	Per Year
Riverside, CA	29.8	2.20	4,261	2,116	1,587	0	0
Los Angeles, CA	25.9	6.45	10,859	4,570	3,019	0	0
Visalia, CA	24.7	0.25	40	16	10	0	0
Fresno, CA	24.0	0.62	967	363	214	0	0
Bakersfield, CA	23.7	0.45	693	254	146	0	0
Orange Co, CA	22.4	1.93	2,810	928	464	0	0
Atlanta, GA	21.2	2.79	3,845	1,124	453	2.4	435
Pittsburgh, PA	21.0	1.60	2,184	624	239	1.8	187
Knoxville, TN	20.4	0.47	623	165	52	2.4	73
Birmingham, AL	20.8	1.24	1,676	467	169	2.4	193
Cleveland, OH	20.3	1.52	2,006	524	158	1.3	128
	Totals		29,964	11,151	6,511		1016
(1) Based on 99/00/01 EPA county level NA list							
(2) Uses CSI 2000-2020 growth of 16.8%							
(3) Conservatively assume entire metro population is exposed to highest county level D.V.							

Table 4
Comparison of Air Pollution-Related Mortality
Estimates and the Leading Causes of Mortality
in 1985 and 2000

Cause of Death	Year & Deaths	
	1985	2000
Total Deaths	2,060,874	2,403,351
Heart Disease	761,714	710,760
Cancer	455,849	553,091
Stroke	151,163	167,661
Lung Disease	73,813	122,009
Heart & Lung Disease	835,527	832,769
Air Pollution Estimate	113,771	110,843
Air Pollution as Percent of Heart and Lung Disease	13.6%	13.3%

FIG 3

Exhibit 18
Sensitivity Analysis: Effect of Thresholds on Estimated 2010 and 2020 Clear Skies Analysis PM-Related Mortality