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Table of Contents
Preface ................................................................. 3
Introduction ........................................................... 4
Benefits and Costs of the Clean Air Act ....................... 5
SIDEBAR: Cost-Benefit Analysis ................................. 6
Assumption I: PM 2.5 Causes Premature Mortality, a/k/a Early Death .............................................. 7
Assumption II: Going to Zero: No Pollutant Threshold Below Which Air is Healthy ......................... 8
Assumption III: Statistical Constructs Equals “Lives Saved” ................................................................. 10
Assumption IV: Co-Benefits of PM 2.5 Reduction Can Justify Any Rule Under the CAA ............................. 11
Conclusion ..................................................................... 15
Endnotes .......................................................................... 17
About the Author ............................................................ 18
Preface

I write this paper on the U.S. Environmental Protection Agency’s (EPA) misuse of science from my six-year former experience as a final regulatory decision-maker for the Texas Commission on Environmental Quality (TCEQ), the world’s second largest environmental regulatory agency after the EPA itself. I was a commissioner and chairman, of TCEQ from 2001-2007. My responsibility for making final decisions on regulations, permits, and enforcement actions necessarily involved my judgments about the rigor, accuracy, and relative uncertainties in diverse scientific studies, statistics, modeling protocols, and technical analyses. I viewed this “science” as a critical tool to inform—but not to dictate—what were ultimately legal and policy decisions.

Various members of the scientific community claim that non-scientists, like me, cannot challenge the credibility of the EPA’s use of science. This view maintains that only credentialed scientists can critique the work of other credentialed scientists. If that is the case, so much the worse for representative democracy.¹ Government by popularly elected representatives on the one hand and government by federal administrators swearing by the authority of science, on the other hand, are contradictory notions. I would call the latter, moreover, an acutely dangerous notion. Regrettably, in the modern United States these two incompatible policy-making models clash often, and with dire results. Elected officials trying to carry out their public duties—e.g. maximizing access to clean, affordable energy—meet stubborn opposition from federal mandarins brandishing their scientific credentials. The magnitude of the EPA’s current regulatory agenda has elevated the importance of these issues.

In my efforts to understand the science on which the EPA grounds its regulatory decision, I am indebted to two notable scientists who have patiently educated me over many years: Dr. Michael Honeycutt, chief toxicologist at TCEQ, and David Schanbacher, P.E., former chief engineer at TCEQ, now director of natural resources for the Texas Comptroller of Public Accounts. I am also grateful for two recent papers which astutely unwind the tangled scientific web now supporting the EPA’s historically “unprecedented regulatory spree.”² My analysis draws heavily on these papers written, respectively, by Dr. Anne Smith of National Economic Research Associates and Dr. Tony Cox, president of Cox Associates.


Introduction

As my late father frequently pointed out (and in a poignant sense proved), “no one gets out of this alive.” Human life is certain to end and is fraught with dangers. Yet life in the 21st century United States is far safer than ever before. Medical science and disease prevention have dramatically reduced, if not eliminated, many disabling and fatal diseases. Life expectancy steadily increases. In highly developed countries like the United States, the most dangerous environmental risks to human life from contaminated water and air have been virtually eliminated.

The U.S. Environmental Protection Agency (EPA), nevertheless, would have Americans believe that hundreds of thousands will die unless its new and unparalleled regulatory agenda is enacted. The EPA undertakes to “protect” us through rules costing many billions of dollars and with cumulative impacts jeopardizing the nation’s electric power supply and millions of jobs. The agency confidently justifies these costs on the value of “preventing deaths” from exposures to a single pollutant rarely considered by physicians to be a killer! The pollutant is known as fine Particulate Matter 2.5 (PM 2.5). See Sidebar: What is Particulate Matter?

After dramatic improvement in air quality and ever-stricter federal air quality standards now approaching natural background levels (see Figure 1), the EPA, in order to justify more stringent regulation, recently devised a method to create a vast reservoir of new health risks. Under the cloak of selective, highly uncertain science driven by implausible assumptions, the EPA now declares that additional regulations are necessary to save thousands of lives. The EPA Administrator Lisa Jackson’s inflammatory claims regularly deceive the public. On “Real Time with Bill Maher,” she grimly warned that “We are actually at the point in many areas of the country … the best advice is don’t go outside. Don’t breathe the air. It might kill you.”

In similarly hyperbolic vein, she told a congressional committee: “If we could reduce particulate matter [pollution] to levels that are healthy, it would have identical impacts to finding a cure for cancer.” This astounding assertion by the head of the EPA demands meaningful explanation. In recent years, cancer has caused the deaths of approximately 600,000 people per year.

This paper aims to demonstrate how several highly questionable assumptions have enabled the EPA to assign health risks at extremely low concentrations of PM 2.5—levels now well

Sidebar: What is Particulate Matter (PM)?

Particulate matter (PM) is a fancy word for natural dust and for the microscopic particles released from man-made activities, especially combustion. PM is everywhere present on the crustal planet earth from natural and man-made sources. To the EPA, particulate matter (PM) is one of the six criteria pollutants regulated under the federal Clean Air Act through National Ambient Air Quality Standards (NAAQS) established by the EPA at a level adequate to protect public health.

PM includes both small solid particles and liquid droplets in the air we breathe. The fine particles in question are minute and measured in microns (micrometers). The width of an average human hair is 70 microns. “Because particles are the byproduct of everything we do in an industrial society as well as natural processes like wind, erosion, forest and brush fires, they are everywhere.” Industrial processes like rock crushing, common domestic activities like cooking, sewing, grilling, wood-burning, combustion of transportation fuels, and farming continually generate PM. Living on a planet composted of dirt, stone, and plants makes PM a ubiquitous component of human life.

The EPA does not distinguish between PM from natural sources such as dirt roads and tilling croplands and PM from urban and industrial sources. Urban PM is likely to be enriched with pollutants with a chemical content potentially more hazardous than natural dust. In spite of many scientific studies stressing this distinction, the EPA still assumes all PM carries the same health risks and regulates accordingly.

The EPA has established a NAAQS for two different sizes of PM: a standard for coarse PM measuring between 2.5 and 10 microns and a standard for fine PM 2.5 microns and lower. The current 24-hour standard for coarse PM 10 is 150 micrograms per cubic meter (ug/m3). The 24-hour standard for PM 2.5 is 35 ug/m3 and the annual standard for PM 2.5 is 15 ug/m3. Although many health-effects studies do not find adverse effects at current levels of PM, the EPA concludes the fine particles (PM 2.5) still pose health risk by irritating or damaging the minute air sacs in the lungs called alveoli. Many toxicological studies, however, find that the natural cleaning system in the lungs removes the minute solids.
below the already precautionary federal standard for PM 2.5. These key assumptions include: 1) Ambient PM 2.5 causes premature death; 2) There is no threshold concentration of ambient PM 2.5 below which risk of premature death ceases; 3) Aggregation of statistical risks is a meaningful surrogate for a human life; and 4) Coincidental reduction of PM 2.5 offers legitimate justification for regulatory initiatives targeting other pollutants.

The EPA is relying almost exclusively on coincidental reduction of PM 2.5 to justify the many new regulations collectively known as the EPA “train-wreck” rules. For example, 99.996 percent of the health benefits supporting the mercury rule derive from coincidental reduction of PM 2.5. Direct reduction of mercury accounts for only 0.004 percent of the rule’s benefits. Without using the inadvertent reduction of PM 2.5 as a hoist, the costs of these new regulations would far surpass their direct benefits. This practice shields the EPA’s rules with few measurable benefits from scrutiny. Further, it subverts the purpose of cost-benefit analysis.

Benefits and Costs of the Clean Air Act from 1990-2020: The Benefits Study

Most of the country already achieves the health-based National Ambient Air Quality Standard (NAAQS) for PM 2.5. Under the federal Clean Air Act, the NAAQS for PM 2.5 and the five other “criteria pollutants” must be set at a level requisite to protect human health with an extra margin of safety and regardless of cost. Thus, the NAAQS are extremely conservative, precautionary standards. “It can be argued that the 1970 Clean Air Act effectively operationalized the absolutist version of the precautionary principle.” Although variously defined, the precautionary principle generally means that with risk of grave, however improbable, harm, and regardless of uncertainty or cost, regulatory intervention is justified.

Since 2009, the EPA has applied a far more precautionary approach than is articulated in the CAA for the health-protective NAAQS. In risk assessments and analyses of the cost and benefits of regulation, the agency no longer regards the ambient pollutant levels set by the NAAQS to be fully protective. The EPA is now attributing risk of premature mortality at PM concentrations approaching and below natural (and thus unpreventable) background levels. Similarly, the EPA is now justifying almost all of its many new air quality regulations on the basis of coincidental reduction of PM 2.5 in rules not intended to address PM 2.5.

This EPA is obsessed with PM 2.5—a criteria pollutant many scientists and regulators believe has already been reduced to healthy levels. To the EPA, however, existing levels of PM 2.5 pose risks to death on a par with cancer! A closer look at an EPA study issued in 2011 reveals the questionable methodology and assumptions behind the EPA’s pre-occupation with
ambient PM 2.5. This study, “Benefits and Costs of the Clean Air Act: Second Prospective Study, 1990-2020,” projects the benefits and the costs of the 1990 amendments to the CAA. The executive summary reveals the EPA’s new methodology. Here the EPA attributes 85 percent of the health benefits projected over the study period (1990-2020) to reduction of ambient levels of PM 2.5. This “Benefits” study finds that CAA regulation will “save” 230,000 lives in 2020. The EPA monetizes the value of those saved lives at nearly $2 trillion but estimates the direct compliance costs at a comparatively paltry $65 billion. The EPA implies that the public pays only $1 dollar for every $30 dollars in health benefits as a result of additional reduction of ambient PM 2.5. Over 90 percent of the $2 trillion derives from alleged prevention of “premature mortality”—roughly equivalent to shortened life expectancy. The EPA further imputes the equivalent of 100 percent certainty to the nearly $2 trillion valuation of the benefits supposed to result from preventing over 230,000 early deaths. “The wide margins by which benefits exceed costs combined with extensive uncertainty analysis suggest it is very unlikely this result would be reversed using any reasonable alternative assumptions of methods.”

If the EPA’s claims about saving lives and gaining trillions of dollars in benefits were factually true, the case for its aggressive regulatory agenda would be compelling. How can society worry about higher electric rates or losing American jobs and businesses to foreign shores when thousands of human lives are at stake? The numbers, however, are so high—such an inflation from previous analyses of PM 2.5 impacts—and so lacking in credible explanation from the EPA that they exceed the bounds of credibility. Peeling back the layers of assumption on which the EPA’s massive benefits depend, one finds that the EPA’s claims are misleading at best, deceptive at worst. What the Benefits study calls an “extensive uncertainty analysis” amounts to an assumption in a cherry-picked model that precludes any other conclusion than a 100 percent probability. Dr. Tony Cox paraphrases the EPA’s claim stating: “Assuming that I am right, it is extremely unlikely that any reasonable combination of alternative assumptions would show that I am wrong.” This is what in logic is called begging the question.

Sidebar: Cost-Benefit Analysis
Cost-benefit analysis, a basic component of Regulatory Impact Analysis (RIA), has long been used to assess the relative advantages or benefits of proposed regulation in comparison to the relative burdens and monetary costs of complying with the regulation. Under an Executive Order issued by President Ronald Reagan in 1981, federal agencies must submit to the White House Office of Management and Budget (OMB) a cost-benefit analysis for all proposed “economically significant” rules. A regulation carrying annual compliance costs of $100 million or more is subject to this requirement. If objectively and comprehensively conducted, cost-benefit analysis should provide key information to regulatory decision makers, elected policymakers, and the public. And while a full RIA should contain a variety of data and analyses, the cost-benefit analysis is a key conclusion. OMB’s current guidance highlights the essential role of cost-benefit analysis in a democracy where regulatory coercion should be the exception and not the rule. “Regulatory analysis is a tool regulatory agencies use to anticipate and evaluate the likely consequences of rules. It provides a formal way of organizing the evidence on the key effects, good and bad, of the various alternatives that should be considered in developing regulations. The motivation is to: 1) learn if the benefits of an action are likely to justify the costs, or 2) discover which of various possible alternatives would be the most cost-effective.”

Under past and present administrations, the EPA has monetized both sides of the cost-benefit equation. The costs are an estimate of the direct costs of compliance incurred by the regulated entity. The benefits typically are an estimate of a dollar-value of the avoidance of morbidity (illness) or premature mortality (shortened life span). The EPA has used diverse methodologies to monetize “work days not lost” or “living longer” but the numbers have become so speculative and inflated as to have no meaningful predictive value.
Assumption I: PM 2.5 Causes Premature Mortality, a/k/a Early Death

The main premise behind the EPA’s promise of massive health benefits from additional regulation is that PM 2.5 causes premature mortality or reduced lifespan. But the selective ecological epidemiological studies upon which the EPA relies to make this claim are incapable of establishing a causal link between death and ambient concentrations of PM 2.5. The two studies on which the EPA relies indicate statistical associations between mortality rates and PM 2.5 concentrations in specific cities. These chronic exposure studies exclude accidental death and somewhat “adjust” for other factors such as smoking or obesity but otherwise attribute all non-accidental deaths to PM 2.5.

The EPA then intricately manipulates the statistical associations through models. The studies can show only an association or a concurrence between slightly elevated mortality rates and PM 2.5 levels. They cannot establish causation. As an example, the statistical correlation between higher rates of swimming and heart attacks in summer months in no way “proves” that swimming causes heart attacks. The correlation between higher incidence of hypothermia and purchase of heavy coats during winter months does not mean heavy coats cause hypothermia.

The EPA’s “Benefits Study” admits that the question of causation is a crucial uncertainty that could lead to “potentially major” overestimation of benefits. “The analysis assumes a causal relationship between PM exposure and premature mortality based on strong epidemiological evidence of a PM/mortality association. However, epidemiological evidence alone cannot establish this causal link.” (Emphasis added.) After acknowledging this uncertainty, the EPA proceeds to the assumption that PM 2.5 causes early death, an assumption made without analyzing the statistical correlations within a causal framework.

Such analytical frameworks exist. Nine analytical criteria, known as the Bradford Hill causal criteria, are widely used by public health scientists to assess whether an observed correlation is or is not likely to be a factual cause. Factors such as biological plausibility and experimental evidence are critical in weighing the health risks from air pollutants. The EPA, on the other hand, imputes complete causal certainty for little reason offered other than the assumption of causation is consistent with current practice. The EPA’s cherry-picked, unvalidated model for the “uncertainty analysis” assigns a probability of 100 percent to the causal connection between PM 2.5 and premature mortality. Such complete certainty is unwarranted by available data and knowledge, as discussed next.

The EPA’s attribution of the equivalent of 100 percent certainty to the assumption that PM 2.5 causes premature mortality also ignores a huge body of credible scientific studies and unanswered questions about which the EPA is certainly aware. The National Academy of Sciences, toxicologists, statisticians and medical doctors have long challenged the findings of epidemiological studies which claim strong evidence of correlations where no causality in fact exists. As Dr. Michael Honeycutt, the chief toxicologist for TCEQ, pointed out in congressional testimony, “Ecological epidemiological studies are not scientifically rigorous to draw conclusions about the cause of health effects identified in the studies … and are not suitable for policy decisions.”

Many confounding variables left unaddressed in the EPA’s selected studies weaken the credibility of even the statistical association, much less the assumption of a causal link between PM 2.5 and premature mortality. Typical confounders include the presence of multiple pollutants co-mingled with PM 2.5 in the ambient air, the diverse composition of PM 2.5 (from natural dust to chemically enriched, and perhaps more hazardous, fine particles) across locations, and the question of whether earlier exposures to PM 2.5 at levels far higher than current levels account for cumulative mortality risks later in life. The current ambient levels of PM 2.5 are far lower than the earlier periods to which subjects of the studies were exposed.

The question of exposure is a major confounder in many of the EPA’s risk assessments. Yet the EPA typically assumes an unrealistic worst-case scenario of maximum exposure 24 hours a day. The EPA’s assumption that all study subjects are equally worst-case scenario of maximum exposure 24 hours a day. The EPA’s assumption that all study subjects are equally exposed to the monitored levels of outdoor PM 2.5 is simply not a representative measure of average, actual exposure. Research shows that PM 2.5 concentrations indoors are much higher than outdoor levels. Yet cleaning the closet, vacuuming, cooking or cruising through a department store can hardly be regarded mortal risks.
The EPA’s estimate of the benefits from reducing PM 2.5-caused morbidity (sickness) also ignores key research data to the contrary. The EPA’s “Benefits” study projects 2.4 million fewer cases of aggravated asthma in 2020. Medical scientists, however, recognize that respiratory infections, mildew, mites, and pet dander more directly exacerbate asthma than ambient air. And incidence of asthma has increased over the past several decades while concentrations of all CAA-regulated pollutants have declined by over 50 percent.

The EPA also disregards studies that show no or even negative correlations. Some studies indicate reduced mortality risks at higher levels of PM 2.5. A recent analysis of mortality risks from PM 2.5 in 27 U.S. communities found a decrease in mortality rates at increased levels of PM 2.5 for one-third of U.S. cities, including Dallas, Houston, Las Vegas and Riverside, California.

Most importantly, the EPA ignores toxicological and clinical studies, which are alone capable of evaluating whether, and to what extent, outdoor concentrations of PM 2.5 may causally impact cardiopulmonary function. Most toxicologists studies contradict the EPA’s PM 2.5 risk assessments. “Toxicological data on typical forms of pollution-derived PM strongly suggest that current ambient concentrations in the U.S. are too small to cause significant disease or death. … The expectation that lives will be saved by reducing ambient PM 2.5 in the U.S. is not supported by the weight of evidence, although other bases for regulating PM may be justifiable.”

**Assumption II: Going to Zero: No Pollutant Threshold Below Which Air is Healthy**

In 2009, the EPA made a methodological change with huge ramifications. The agency now calculates mortality risks from PM 2.5 below the health protective level of the NAAQS (presently set at an annual 15 ug/m3). It also calculates them below the lowest measured ambient level (LML) in the original studies and even below natural background levels approaching zero. Remarkably, the EPA now assumes that there is no level of PM 2.5 below which risks to premature death cease. Statisticians call this a “no threshold linear regression to zero analytic model.” In laymen’s terms, no risk is too low.

Prior to 2009, the EPA did not estimate risks below the lowest ambient level measured in the epidemiological studies. If the PM level in a given location was already below the LML (typically 10 ug/m3), the agency did not assume additional reductions in PM 2.5 would generate additional health benefits. “However, starting in 2009, the EPA decided that it would calculate risks to the lowest level projected by its air quality models, even though no observed or empirical evidence exists … in that low concentration zone.”

The statistical associations between premature mortality and PM 2.5 identified in the epidemiological studies cease below the lowest measured level in the study. But the EPA now imputes, by extrapolation, the same risks (and at the same rate) for PM 2.5 levels for which no statistical evidence exists. “Extrapolation is the use of quantitative relationships outside the range of evidence on which it was based.”

The EPA’s adoption of this no-threshold approach to assessing risk increased by almost four-fold. The EPA’s estimate of total U.S. deaths attributable to PM 2.5 pollution—from 88,000 to 320,000! This approach means, according to the EPA at least, that over two-thirds of the public’s health risk from exposure to PM 2.5 comes from ambient levels not only far below the protective national standards known as the NAAQS but even below the lowest modeled levels in the relevant studies.

In short, the EPA’s incredible finding is that mortal risks increase in proportion to the extent that a location’s ambient concentration of PM 2.5 exceeds natural background levels—now estimated by the EPA at the extremely low figure of 1 ug/m3. “This created a major change in the level of national mortality estimated to be due to PM 2.5 because the majority of the U.S. population resides in locations where the ambient PM 2.5 concentrations are below 10ug/m3.”

Despite critical questions from members of Congress, senior EPA leadership recently defended adoption of the no-threshold approach. Says Gina McCarthy, assistant administrator of the EPA: “Studies demonstrate an association between premature mortality and fine particle pollution at the lowest levels measured in the relevant studies, levels that are significantly below the NAAQS for fine particles. These studies have not observed a level below which premature
mortality effects do not occur. The best scientific evidence ... is that there is no threshold level of fine particle pollution below which health risk reductions are not achieved by reduced exposure.” This is another way of saying: No risk is too low, improbable, or uncertain that it is not worth regulating.28

The EPA claims that the two studies in question show no evidence of a threshold, but many studies ignored by the EPA do show a threshold. The agency’s Benefit Study admits that the “no-threshold” assumption is a “key uncertainty” but as usual assigns a “high” confidence to the model that incorporates this assumption. The single study that the EPA cites to support this questionable “no-threshold” assumption is one funded by its own Health Effects Institute. And importantly, the “no-threshold” assumption violates the foundational principle of toxicology. It is the dose that makes the poison. The EPA’s defense of this absurdly precautionary assumption is another way of saying that the point at which all risk is zero cannot be proven. This is not surprising. How can any negative proposition be proven with complete certainty?

The EPA also maintains that its adoption of a “no-threshold” assumption in 2009 was endorsed by the agency’s various scientific advisory panels. The growing evidence of financial conflicts of interest among the members of the EPA’s technical review panels casts doubts on the objectivity of these review panels. Six of the seven members of the EPA’s Clean Air Science Advisory Committee (CASAC) have received EPA grants to conduct research for the agency.29 CASAC Chairman Jonathan Samet was the principal researcher for grants of $9.5 million dollars. The EPA’s inspector general has begun an investigation of these alleged conflicts of interest.30

Figure 2: Risk Attributed to Ambient PM 2.5

![Graph showing the percentage of avoided PM-related deaths at different baseline annual mean PM$_{2.5}$ levels]

Source: Table 5-15, EPA’s RIA in final Utility MACT (mercury) Rule.
Lives saved, deaths prevented or avoided, and premature mortality: the EPA's terms are misleadingly imprecise. “Avoided deaths” do not occur since clean air does not confer immortality.

Nor, despite extremely low concentrations of PM 2.5 in most areas of the country, did the EPA give any public notice of the regulatory implications of this sea-change in risk assessment of current air quality conditions. Public health scientists may have long debated the relative merits of no-threshold linear regression analysis, but these were scientific debates without the economic and societal implications at stake in the EPA’s regulatory agenda, unprecedented in its cumulative impacts.

A growing number of policy makers, state agencies, scientists, physicians and concerned voters are baffled by the EPA’s inflated claims about low levels of PM 2.5. Public disclosure of the data behind the EPA’s claim has not been forthcoming even after repeated congressional requests. U.S. Rep. Andy Harris (R-MD), a medical doctor who chairs the Energy and Environment subcommittee of the House Science, Space and Technology Committee, typifies growing frustration with the lack of transparency in the EPA’s science. “If our current air,” he has said, “is such a threat to human health that it is killing hundreds of thousands of people each year, I am very interested to review the information the agency relies on in establishing this relationship … Because the EPA is not transparent with the sources of their data … EPA seems to rely on making statistical hay out of minor associations between pollutants and premature mortality.”

Assumption III: Statistical Constructs = “Lives Saved”

The EPA’s public pronouncements trumpet the dire need for additional regulation to save thousands of lives. Such unequivocal, emotional pronouncements grossly mislead the public and can intimidate even the hardened skeptic. A headline on the summary for the EPA’s “Benefits” study is typical: “In 2020, the CAA Amendments will prevent over 230,000 early deaths.” Administrator Jackson regularly tells the media the Clean Air Act has saved “literally hundreds of thousands of lives,” or “public health protections will mean the difference between … life and death for hundreds of thousands of citizens.”

These “saved lives” are nothing more than statistical constructs; they do not refer to real people. When not speaking for public consumption, the EPA calls them “statistical lives.” For the thousands of lives that the EPA claims air pollution has ended or that CAA regulation will save, there is not one identified individual. Nor are there specific medical conditions or causes of death attributed to PM 2.5 exposures. The EPA’s typical approach is to assume any non-accidental death from cardiopulmonary conditions is caused by air quality.

Lives saved, deaths prevented or avoided, and premature mortality: the EPA’s terms are misleadingly imprecise. “Avoided deaths” do not occur since clean air does not confer immortality. The health benefits the EPA projects from regulatory reduction of PM 2.5 is more accurately described as reduction in the relative risk of mortality. Extended life-expectancy or life-years gained more accurately describe the health benefit at issue.

The EPA constructs a “statistical life” (SL) by measuring the reduction in statistical risks assumed to result from reduction of ambient PM 2.5. “A ‘statistical life’ has traditionally referred to the aggregation of small risk reductions to many individuals until that aggregate reflects a total of one statistical life.” Quite obviously, “statistical lives saved” bear no relationship to actual individual human lives. The nearly $2 trillion monetary value of “preventing 230,000 deaths” in the Benefits Study derives from a simple calculation. The EPA monetizes the value of one statistical life at $8.9 million.

Thus: 230,000 “prevented deaths” x $8.9 million per statistical life saved = $1.8 trillion.

The EPA’s valuation of one statistical life at $8.9 million is dubious. The EPA’s favored studies find that the median age of people who gain additional life expectancy is 80 years. And the increased life expectancy is estimated in several months, not years. But when aggregated into one statistical life, the EPA sets a value of $8.9 million per statistical
The monetized value of additional life expectancy for an 80-year old is typically estimated at about one-sixth the value of an individual 25 years old. Thus, if a more regularly used value for the octogenarian is used, the benefits decline by six-fold. 

Thus: 230,000 “prevented deaths” x 1/6 of $8.9 million = $300 billion (instead of $2 trillion).

And if the factual accuracy of the EPA’s three key assumptions is assigned a probability of 50 percent rather than 100 percent, the costs of regulatory reduction of PM 2.5 dwarfs the projected health benefits with a ratio of $65 billion (costs) to $19 billion (benefits). With the more plausible assumption of 50 percent probability, the estimated health benefits fall from almost $2 trillion to $19 billion. (See Figure 3). The EPA’s dramatic claims are highly sensitive to the unjustified certainty ascribed to the assumptions. “The EPA’s evaluation of health benefits is unrealistically high, by a factor that could well exceed 1,000 and that it is therefore very likely that the costs of the 1990 CAA exceed its benefits, plausibly by more than 50-fold.”

Assumption IV: Co-Benefits of PM 2.5 Reduction Can Justify Any Rule Under the CAA.

The EPA is now supporting new air quality regulations imposing multi-billion dollar costs on the basis of alleged mortality risks from trace levels of PM 2.5 created by the “no-threshold” approach. The EPA increasingly uses these “coincidental reductions” of PM 2.5 to justify the benefits of regulations intended to control not PM 2.5 but different pollutants such as mercury, ozone, and sulfur dioxide. The EPA’s cost-benefit analysis calls these coincidentally occurring reductions “co-benefits.”

This practice of relying on “co-benefits” from PM 2.5, evidently started in 1997 when the EPA issued the first NAAQS for PM 2.5. Since 2009, however, the EPA has increasingly used PM 2.5 co-benefits as the primary, if not exclusive, source of health benefits in rulemakings under the Clean Air Act directed to other pollutants. As examples, the EPA’s mercury rule, industrial boiler rules, and the new SO2 NAAQS rely on co-benefits from PM 2.5 reduction for over 99 percent of estimated health benefits. Without these co-benefits, the EPA’s regulatory analysis of direct costs of these rules would far exceed any measurable benefits.

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Figure 3: Health Benefits from PM 2.5 Reduction with Alternative Assumptions

<table>
<thead>
<tr>
<th>Assumption</th>
<th>Description</th>
</tr>
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<tbody>
<tr>
<td>1.</td>
<td>One-sixth reduction of the EPA’s $8.9 million for VSL/VSLY for median age of 80 year old</td>
</tr>
<tr>
<td>2.</td>
<td>50% probability (instead of 10%) that assumption of true association: PM2.5 and premature death</td>
</tr>
<tr>
<td>3.</td>
<td>50% probability (instead of 10%) that association is causal</td>
</tr>
<tr>
<td>4.</td>
<td>50% probability (instead of 10%) of no PM2.5 threshold ambient health effects cease</td>
</tr>
<tr>
<td>5.</td>
<td>50% probability of reduction of health effects due to disease prevention &amp; medication</td>
</tr>
</tbody>
</table>

Benefits Study: 230,000 SL X $8.9 million (per VSL) = $1.8 trillion benefits. Costs=$65 billion

With alternative assumptions above: Health Benefits = $19 billion. Costs = $65 billion

($1.8 trillion x(1/6)x(0.5)x(0.5)x(0.5)x(0.5) = $19 billion

The EPA’s “no-threshold” assumption in 2009 vastly increased the benefits that the EPA could ascribe to coincidental reduction of PM 2.5 in regulations not targeting this pollutant.

The EPA admits that the direct health benefits from reduction of mercury account for only 0.004 percent (or $6 million) of the health benefits. And the PM 2.5 co-benefits account for 99.996 percent of what the EPA values as $140 billion in health benefits. The EPA estimates the direct costs of the rule at $11 billion. The agency’s press releases and congressional testimony do not acknowledge this huge gap between direct mercury benefits and indirect PM 2.5 benefits, but the Federal Register notice for this rule explicitly reveals the glaring gap.38

Dr. Anne Smith of National Economic Research Associates (NERA) has completed a thoroughly researched analysis of the EPA’s use of co-benefits in “An Evaluation of the PM 2.5 Health Benefits Estimates in Regulatory Impact Analyses for Recent Air,” a work from which this present paper draws heavily.39 Dr. Smith analyzed the Regulatory Impact Analyses (RIA) for over 50 CAA-related rules promulgated since 1997. (See Sidebar: Cost Benefit Analysis).

As shown in Figure 4 (next page), Dr. Smith found a growing reliance on co-benefits from PM 2.5 reductions. In 13 RIAs for rules not targeting PM 2.5, submitted between 2009-2011, co-benefits from PM 2.5 accounted for more than half of all estimated health benefits. In six of the cost-benefit analyses, co-benefits from PM 2.5 accounted for 100 percent of the benefits.

The EPA’s “no-threshold” assumption in 2009 vastly increased the benefits that the EPA could ascribe to coincidental reduction of PM 2.5 in regulations not targeting this pollutant. As depicted in Figure 2, 94 percent of the 11,000 (statistical) lives purportedly “saved” by the mercury rule derive from PM 2.5 co-benefits in geographical areas that already attain the current PM 2.5 NAAQS of 15 ug/m3. Recall that NAAQS are conservative federal standards below which human health should be fully protected. The EPA’s increasing reliance on co-benefits garnered from PM concentrations approaching background levels is an evasion of the EPA’s fundamental responsibility under the CAA to directly regulate the criteria pollutants, of which PM 2.5 is one.

By relying on co-benefits from PM 2.5, the EPA also evades its obligation to justify the need for stricter regulations. Without the 99.9 percent plus co-benefits from PM 2.5, the EPA’s case for the health benefits supposedly obtained under the recently issued National Emission Standards for Hazardous Air Pollutants (NESHAP) would evaporate. Consider also the mercury rule, acknowledged by the EPA to be the most expensive CAA regulation to date, and widely viewed as a threat to electric reliability. The rule is based on PM 2.5 co-benefits in areas now attaining the NAAQs. It becomes on these grounds a disservice to the public, to policy makers and not least to the many employees whose job may end as a result of this regulation.

If the EPA is convinced that ambient PM 2.5 now presents dire health risks, the agency should make its case for strengthening the PM 2.5 NAAQS. The EPA is now reviewing the current 15 ug/m3 NAAQS PM 2.5 and apparently may reduce that standard to a level within a range of 10 to 13 ug/m3 or lower. Co-benefits from another pollutant should not be used in a cost-benefit analysis to justify regulation of another pollutant.

“Clearly, EPA’s PM 2.5 co-benefits habit is allowing EPA to avoid grappling with the important task of making a case that all of these other pollutants really require tighter controls. … but a high degree of complacency and analytical laziness has instead taken root … The situation is completely at odds with the purpose of RIAs, which is to provide a consistent, credible and thoughtful evaluation of the societal value gained with increased regulatory burden that new rulemakings create.”40

“In all, EPA’s use of co-benefits should end for several reasons. It scares the public into believing that large numbers of people [would] die prematurely were it not for implementation of new rules on pollutants for which EPA has not actually identified any current public health risk.”41
## Figure 4: Degree of Reliance on PM 2.5-Related Co-Benefits in RIAs

<table>
<thead>
<tr>
<th>Year</th>
<th>RIAs for Rules Not Targeting Ambient PM 2.5</th>
<th>PM Co-Benefits Are &gt;50% of Total</th>
<th>PM Co-Benefits Are Only Benefits Quantified</th>
</tr>
</thead>
<tbody>
<tr>
<td>1997</td>
<td>Ozone NAAQS (.12 1hr=&gt;.08 8hr)</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>1997</td>
<td>Pulp &amp; Paper NESHAP</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1998</td>
<td>NOx SIP Call &amp; Section 126 Petitions</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>1999</td>
<td>Regional Haze Rule</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>1999</td>
<td>Final Section 126 Petition Rule</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>2004</td>
<td>Stationary Reciprocating Internal Combustion Engine NESHAP</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>2004</td>
<td>Industrial Boilers &amp; Process Heaters NESHAP</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>2005</td>
<td>Clean Air Mercury Rule</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>2005</td>
<td>Clean Air Visibility Rule/BART Guidelines</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>2006</td>
<td>Stationary Compression Ignition Internal Combustion Engine NSPS</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>2007</td>
<td>Control of HAP from Mobile Sources</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>2008</td>
<td>Ozone NAAQS (.08 8hr=&gt; .075 8hr)</td>
<td>X</td>
<td></td>
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<tr>
<td>2008</td>
<td>Lead (Pb) NAAQS</td>
<td>X</td>
<td></td>
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<tr>
<td>2009</td>
<td>New Marine Compression Ignition Engines &gt; 30 L per Cylinder</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>2010</td>
<td>Reciprocating Internal Combustion Engines NESHAP -- Compression Ignation</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>2010</td>
<td>EPA/NHTSA Joint Light-Duty GHG &amp; CAFES</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>2010</td>
<td>SO2 NAAQS (1-hr, 75 ppb)</td>
<td>X</td>
<td>&gt; 99.9%</td>
</tr>
<tr>
<td>2010</td>
<td>Existing Stationary Compression Ignition Engines NESHAP</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>2011</td>
<td>Industrial, Commercial, and Institutional Boilers NESHAP</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>2011</td>
<td>Industrial, Commercial, and Institutional Boilers &amp; Process Heaters NESHAP</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>2011</td>
<td>Commercial &amp; Industrial Solid Waste Incin. Units NSPS &amp; Emission Guidelines</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>2011</td>
<td>Control of GHG from Medium &amp; Heavy-Duty Vehicles</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>2011</td>
<td>Ozone Reconsideration NAAQS</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>2011</td>
<td>Utility Boiler MACT NESHAP (Final Rule’s RIA)</td>
<td>X</td>
<td>≥ 99%</td>
</tr>
<tr>
<td>2011</td>
<td>Mercury Cell Chlor Alkali Plant Mercury Emissions NESHAP</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>2011</td>
<td>Sewage Sludge Incineration Units NSPS &amp; Emission Guidelines</td>
<td>X</td>
<td>X</td>
</tr>
</tbody>
</table>

*Source: A. Smith, Co-Benefits, p.18.*
Figure 5: Business Impacts

Note: Percentage change in productivity under the CAA for the year 2020. EPA estimate by EMPAX-CGE model.
Source: EPA Benefits Study, p. 8-20
Figure 6: Health Effects of Poverty and Unemployment

The macro level analysis has demonstrated the fundamental importance of economic growth as a central source of mortality reduction, whereas economic decline—especially in the form of high unemployment—should increase morbidity and mortality rates, whereas economic status improvement, should lead to lower morbidity and mortality. It is now among the firmest of epidemiological findings, across industrialized societies, that socioeconomic status is inversely related to health status. In particular, higher income has been repeatedly shown at the national level that mortality rates, whereas economic decline—especially in the form of high unemployment—should increase morbidity and mortality rates, whereas economic status improvement, should lead to lower morbidity and mortality. It is now among the firmest of epidemiological findings, across industrialized societies, that socioeconomic status is inversely related to health status.

Conclusion

Many reputable scientific bodies have severely criticized the weakness of the science the EPA now relies upon to justify new rules. Among these critics: the National Academy of Science and the National Research Service, along with the EPA’s own Scientific Advisory Board, Board of Scientific Counselors, and Clean Air Act Advisory Council. Dr. Thomas Burke, chairman of a recent National Academy of Science (NAS) review panel on the EPA’s chemical risk assessment, told EPA officials that “EPA science is on the rocks … if you fail, you become irrelevant, and that is kind of a crisis.” The EPA’s chemical risk assessment for formaldehyde set the level for adverse health effects—and thus regulations—several times lower than the average natural level of formaldehyde in human exhalation.

Current EPA science has a pattern. The agency relies on one or two cherry-picked studies which indicate the most adverse health effects at the lowest concentration of the pollutant in question. The EPA either ignores or gives lip service to sometimes hundreds of equally reputable studies that contradict these studies. The EPA’s favored studies are usually ecological epidemiological studies that show intricately manipulated statistical associations rather than data-driven causal connections between pollutant levels and adverse health effects. And instead of characterizing the relative uncertainties in the scientific studies on which the EPA relies, and weighing the evidence from diverse studies, the EPA publicly declares complete certainty and approval by peer review. Upon a closer look, the peer-reviewers regularly are either EPA employees, scientists who wrote the relevant studies or were employed by the same institution which the EPA paid to conduct the study.

The EPA would have the public believe that “pure science” shows that a fossil-fuel supplanting agenda is necessary to save the lives of hundreds of thousands. Note in Figure 5, the EPA’s Benefits Study projects the decline of fossil fuel based industry as well as the energy intensive manufacturing and chemical industries dependent on affordable, efficient fossil fuels.
Sound science and objective scientists abound. Science in the hands of government, however, is easily compromised in order to reach predetermined policy outcomes.

Environmental regulatory standards reflect a judgment about what is acceptable or unacceptable societal risk. As such, the EPA’s final regulatory decisions are ultimately policy decisions that no scientific findings can dictate. The EPA’s manipulation of cost-benefit analyses to project massive benefits at comparatively modest cost denies policymakers and the public the information needed to weigh the many trade-offs involved in complex societal decisions about unacceptable risks. Economic impact does matter, and it matters to health. Many studies show that income and employment strongly correlate with health and life span. *(See Figure 6)*

Sound science and objective scientists abound. Science in the hands of government, however, is easily compromised in order to reach predetermined policy outcomes. If the current EPA’s policy objective is to supplant fossil fuels, PM 2.5 is a useful tool. PM 2.5 is an ever-present byproduct of combustion of coal, natural gas, and oil. Emissions from cars and trucks, however, have been reduced by over 90 percent, at the same time vehicle miles traveled increased by 165 percent. Natural processes will always release fine particles into the ambient atmosphere of this planet.

The EPA’s science is, indeed, on the rocks, as the chairman of the NAS review concluded. The Clean Air Act under which the EPA conducts risk assessment and sets national standards needs to stipulate minimal criteria for scientific risk assessment of health effects, sufficiently robust to guide decisions on air quality standards. Such minimal criteria would include the following:

- The EPA’s risk assessments must be peer-reviewed by an independent body—not, as now, within the agency itself.
- Toxicological studies and clinical trials demonstrating causal connections between ambient levels of a pollutant and adverse health effects trump epidemiological studies indicating statistical correlations. Ecological epidemiological studies, alone, are not rigorous enough to set national ambient or emission standards.
- Abandonment of no threshold linear regression modeling assumptions in setting ambient standard or regulatory emission limits.
- Health-based air quality standards that incorporate representative estimates of actual exposure and not the implausible assumption of 24-hour exposure to the highest monitored level.
- Physical measurement through monitored readings trump models.
- A plausible biological mechanism as predicate for health-effects findings.
- Comprehensive, cumulative cost-benefit analysis of all rules according to methodology and scope stipulated in law.

The EPA’s regulatory sway is at a tipping point. Existing technologies cannot meet the EPA’s new emission limits unless this country overnight can replace 85 percent of the energy on which our current way of life relies. Short of a miraculous breakthrough in technology, the EPA’s regulatory agenda is a perilous pipe-dream precluded by the laws of math and physics.
Endnotes

10. Ibid.
17. Ibid, supra, note 11: 5.
19. Susan Hildebrand, PE, Chief Engineer, Michael Honeycutt, Ph.D., Stephanie Shirley, Ph.D., "EPA's Benefit Cost Analysis" (4 Apr. 2012).
25. Ibid.
27. Ibid.
30. Hayward, supra, note 21.
40. Ibid, 15.
41. Ibid, 33.
42. "Key Advisor Warns EPA to Improve Agency Science or Face a Crisis," *InsideEPA.com* (8 July 2011).
43. Michael Honeycutt, Ph.D., Texas Commission on Environmental Quality, Comments Regarding the Use of Science in, and Implications of, EPA's Chemical Risk Assessments (4 Oct. 2011).
44. Ibid.
45. Hayward, supra note 21.
About the Author

Kathleen Hartnett White joined the Texas Public Policy Foundation in January 2008. She is a Distinguished Senior Fellow-in-Residence and Director of the Armstrong Center for Energy & the Environment.

Prior to joining the Foundation, White served a six-year term as Chairman and Commissioner of the Texas Commission on Environmental Quality (TCEQ). With regulatory jurisdiction over air quality, water quality, water rights & utilities, storage and disposal of waste, TCEQ’s staff of 3,000, annual budget of more than $600 million, and 16 regional offices make it the second largest environmental regulatory agency in the world after the U.S. Environmental Protection Agency.

Prior to Governor Rick Perry’s appointment of White to the TCEQ in 2001, she served as then Governor George Bush appointee to the Texas Water Development Board where she sat until appointed to TCEQ. She also served on the Texas Economic Development Commission and the Environmental Flows Study Commission. She is now serving in her fifth gubernatorial appointment as an officer and director of the Lower Colorado River Authority.

White is also co-owner of White Herefords and a partner with her husband in a 125 year-old ranching operation in Jeff Davis and Presidio counties. She also is Vice-Chairman of the Texas Water Foundation and sits on the board of the Texas Natural Resource Foundation. She recently received the 2007 Texas Water Conservation Association’s President’s award, the Colorado River Foundation’s Friend of the River Award and the Texas Chemical Council’s Leadership Award.

A writer and consultant on environmental laws, free market natural resource policy, private property rights, and ranching history, White received her bachelor cum laude and master degrees from Stanford University where for three years she held the Elizabeth Wheeler Lyman Scholarship for an Outstanding Woman in the Humanities. She was also awarded a Danforth National Fellowship for doctoral work at Princeton University in Comparative Religion and there won the Jonathan Edwards Award for Academic Excellence. She also studied law under a Lineberry Foundation Fellowship at Tech University.

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