The EPA's Pretense of Science

Regulating Phantom Risks

December 2019

by Hon. Kathleen Hartnett White
Brent Bennett, Ph.D.

Texas Public Policy Foundation

December 2019

by Hon. Kathleen Hartnett White
Brent Bennett, Ph.D.

Armstrong Center for Energy
& the Environment

Texas Public Policy Foundation

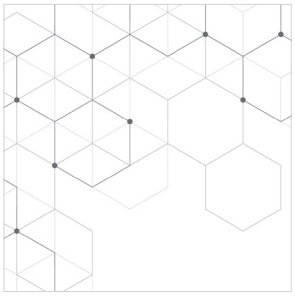


Table of Contents

Executive Summary....3

Introduction....3

What is Particulate Matter?....4

Cost-Benefit Analysis in the Regulatory Process....6

EPA's Study of Benefits and Costs of the Clean Air Act From 1990-2020 6

Assumption I: PM2.5 Causes Premature Mortality, aka Early Death 7

Assumption II: No Pollutant Threshold Below Which Air Is Healthy....8

Assumption III: Statistical Constructs Equals "Lives Saved" 9

Assumption IV: Co-Benefits of PM2.5 Reduction Can Justify Any Rule Under the CAA....10

Recommendations....12

Conclusion....13

References....14

The EPA's Pretense of Science
Regulating Phantom Risks

by Hon. Kathleen Hartnett White and Brent Bennett, Ph.D.

Executive Summary

Among the six criteria pollutants stipulated in the Clean Air Act (CAA), fine particulate matter (PM2.5) and ground-level ozone (O_3) have generated the most attention and debate over the past two decades. This debate reached a fever pitch under the Obama administration, as the Environmental Protection Agency (EPA) conducted a "regulatory spree" that, according to the *Wall Street Journal*, was unprecedented in its scope, speed, and stringency, and colossal in costs (WSJ Editorial Board). Stricter regulations on mercury, carbon dioxide, and other types of air emissions—costing billions of dollars and risking the loss of millions of jobs—were primarily justified by huge benefits from the reduction of PM2.5.

The EPA created these estimated benefits by applying a "linear no-threshold" model that found public health benefits of reducing PM2.5 all the way down to zero, even below the natural background levels of dust in the air in most places around the U.S. This model has been used despite the fact that the National Ambient Air Quality Standard (NAAQS) for PM2.5 is set at an annual average of 12 micrograms per cubic meter ($\mu g/m^3$), which the CAA says should be a level which is "requisite to protect the public health" with "an adequate margin of safety" (United States Code).

The current EPA is now engaged in reforming the flawed risk assessments and cost-benefit analyses for PM2.5. Even as the current EPA works to reform these abuses, there are still many flaws in the quantification of benefits attributed to reductions in PM2.5 levels. The causal link between PM2.5 and premature mortality at low concentrations is still a subject of debate and needs to be established before any benefits can be attributed to reducing ambient PM2.5 below current levels. In addition, no thresholds have been established to reflect the fact that reducing PM2.5 levels to zero is both practically impossible and not of any benefit to public health. Finally, the method the EPA uses to quantify the reduction in mortality from reducing PM2.5 and ascribes a monetary value to that reduction needs serious reform.

This paper explains some of these flaws, highlights how they were abused by the Obama-era EPA, and recommends reforms that should be implemented by the current EPA and ultimately enacted into an updated CAA. We conclude that the current PM2.5 NAAQS should not be tightened any further below 12 $\mu g/m^3$, as the costs would far outweigh any potential benefits.

Key Points

- The Obama-era EPA inflated the benefits of a wide array of new air quality regulations with supposed health benefits from reducing PM2.5 below the already safe standard of 12 ug/m3.
- Despite toxicological evidence that humans have a natural resistance to PM2.5, the EPA has consistently applied a "no-threshold" assumption and said that there is no level of pollution that is too low to prevent harm. This assumption should be rigorously examined using toxicological studies and abandoned if not upheld in that field.
- The EPA needs better toxicological studies and clinical trials demonstrating causal connections between ambient levels of PM2.5 and adverse health effects. Epidemiological studies relying on statistical correlations are not rigorous enough to justify setting new air quality standards.
- The EPA needs to justify lowering the standard for PM2.5 before it can claim co-benefits from lowering PM2.5 levels through other regulations.

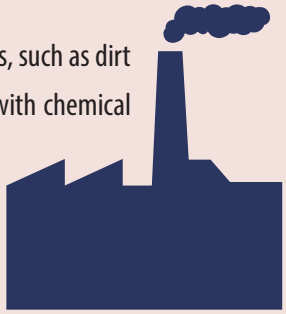
Introduction

Deep within the Environmental Protection Agency (EPA), well below the radar of the general public, an important debate is taking place about how to differentiate scientific and policy considerations in the formulation of environmental regulations. This debate has been ongoing for many years, but it has reached a new level as the EPA under the Trump administration takes on reform of the rulemaking process and more clearly delineates these considerations (Pruitt, 10). Because of the broad reach of environmental regulations, this debate has far

WHAT IS PARTICULATE MATTER?

Particulate matter (PM) is a fancy word for natural dust and 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. For the EPA, PM is one of the six criteria pollutants regulated under the federal Clean Air Act (CAA) through National Ambient Air Quality Standards (NAAQS). The EPA establishes the standards at a level requisite to protect the public health with a margin of safety (United States Code).

PM includes both small solid particles and liquid droplets and is present 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 industrialized society, as well as natural processes like wind, erosion, forest and brush fires, they are everywhere" (Ropeik and Gray, 169). Industrial processes like rock crushing, common domestic activities like cooking, grilling, wood-burning, combustion of transportation fuels, and farming generate PM continually. Living on a planet composed of dirt, stone, and plants makes PM a ubiquitous component of human life.



Because of the wide variety of sources of PM, it is important to distinguish between PM from natural sources, such as dirt roads and tilled cropland, and PM from urban and industrial sources. Urban PM is more likely to be enriched with chemical content potentially more hazardous than natural dust. The CAA dictates that the EPA administrator be advised on the relative contribution "of natural as well as anthropogenic activity" (United States Code). However, there is no specific requirement that this distinction be incorporated into the NAAQS, and the EPA has only set single standards for PM2.5 with no accounting for regional differences in natural background levels or international transport (CFR 2013a, § 50.18).

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 PM10 is 150 micrograms per cubic meter ($\mu g/m^3$). The 24-hour standard for PM2.5 is 35 $\mu g/m^3$, and the annual standard for PM2.5 is 12 $\mu g/m^3$ (CFR 2013a, § 50.18). Although many health effects studies do not find adverse effects at current levels of PM, the past EPA concluded the fine particles (PM2.5) still pose health risks by irritating or damaging the minute air sacs in the lungs called alveoli. Many toxicological studies (Green and Armstrong; Koop and Tole 2004; Koop and Tole 2006), however, find that the natural cleaning system in the lungs removes the minute solids.

reaching implications. In the case of air quality regulations under the Clean Air Act (CAA), billions of dollars and millions of jobs are weighed against the benefits of reduced emissions of air pollutants.

The central issues in this debate are to what extent scientists and their beliefs should dictate the outcome of the rule-making process—both within the EPA and in court cases—and how much authority our elected representatives should delegate to the administrative branches like the EPA.

Many members of the scientific community claim that if credentialed scientific advisors offer a certain view of the world, a certain set of policy decisions should follow.[1] This model of policymaking, frequently adopted by the Obama-era EPA, elevated science beyond its proper role as a critical tool to inform—but not to dictate—what are ultimately legal and policy decisions in a representative democracy. Those decisions should be made by elected representatives, who are directly accountable to the people whom they serve and regulate.

1 Co-author Kathleen White addresses the technical subject of air pollution regulations as a former (2001-2007) chairman and commissioner for the Texas Commission on Environmental Quality (TCEQ), the second-largest environmental regulatory agency in the country after the EPA itself. Her responsibility for making final decisions on regulations, permits, and enforcement actions necessarily involved judgments about the rigor, accuracy, and relative uncertainties in diverse scientific studies, modeling protocols, and technical analyses. Co-author Brent Bennett, Ph.D., approaches these issues as a credentialed materials scientist with a broad scientific background but without specific expertise in the fields most pertinent to air pollution regulations, such as atmospheric science and toxicology.

Regrettably, in the past several decades, these two incompatible policymaking models often clashed because the U.S. Congress has delegated far too much lawmaking power to the administrative apparatus. This situation is especially true for the CAA, which gives the EPA the authority to set regulations "requisite to protect the public health" with "an adequate margin of safety" but does not stipulate that the EPA must consider the cost of the regulations or their economic impact (United States Code).

Among the many pollutants covered by the CAA, fine particulate matter (PM2.5) and ground-level ozone have become the most controversial in recent years. Regarding PM2.5, the Obama-era EPA regularly told the American public that this pollutant was one of the greatest causes of death in the U.S. For example, on *Real Time with Bill Maher*, former EPA Administrator Lisa Jackson grimly warned, "We're actually at the point in many areas of this country where on a hot summer day, the best advice we can give you is don't go outside. Don't breathe the air, it might kill you" (quoted in Reis). In a similarly hyperbolic vein, she told a congressional committee, "If we could reduce particulate matter to healthy levels, it would have the same impact as finding a cure for cancer in our country" (Jackson 2011a, 56).

These assertions by a former head of the EPA demand a meaningful explanation. The Centers for Disease Control and Prevention (CDC) says cancer causes the deaths of approximately 600,000 people per year (CDC 2017a). CDC data indicates that the leading cause of death in the U.S. is heart disease, followed by cancer, and then injuries. Chronic lower respiratory diseases are the fourth leading cause of death (CDC 2017b), killing more than 160,000 people each year, and smoking accounts for 74 percent of those deaths, not air pollution (CDC 2018a).

Nevertheless, the Obama-era EPA told Americans that hundreds of thousands would die unless an unparalleled regulatory agenda was enacted. The EPA undertook to "protect" us through rules costing many billions of dollars and with cumulative impacts jeopardizing the nation's power supply and millions of jobs, confidently justifying these costs on the value of "preventing deaths" from exposure to PM2.5. In spite of the dramatic improvements in air quality and ever-stricter federal air quality standards now approaching natural background levels (see **Figure 1**), the Obama EPA utilized a methodology that increased premature mortality by fourfold, from 88,000 to 320,000. Under the cloak of selective, highly uncertain science driven by implausible assumptions, the EPA then declared that additional regulations were necessary to save thousands of lives.

Several questionable assumptions have enabled the past EPA to assign health risks at extremely low concentrations of PM2.5—levels now well below the already precautionary federal standard for PM2.5. Key assumptions made include (1) ambient PM2.5 causes premature death; (2) there is no threshold concentration of ambient PM2.5 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 PM2.5 offers legitimate justification for regulatory initiatives targeting other pollutants.

The Obama EPA relied almost exclusively on coincidental reduction or co-benefits of PM2.5 to justify the many new regulations collectively known as the EPA "train-wreck" rules (White, 10). For example, 99.993 percent of the health benefits supporting a rule to reduce mercury derived from the coincidental reduction of PM2.5 (Federal Register 77, 9306). Direct reduction of mercury accounted for only 0.007 percent of the rule's benefits (9306). Without using the "co-benefit" of reducing PM2.5 as a hoist, the costs of these new regulations would far surpass the direct benefits.

Figure 1. Air quality improvements since 1980

	Ambient 1980- 2018	Emissions 1980- 2018
Carbon Monoxide (CO)	-83%	-73%
Ozone (O_3)	-31%	N/A
Lead (Pb)	-99%	-99%
Nitrogen Dioxide (NO_2)	-65%	-62%
Particulates (PM10)*	-26%	-25%
Fine Particulates (PM2.5)**	-39%	-37%
Sulfur Dioxide (SO_2)	-91%	-90%

N/A – Ozone is a by-product that is not directly emitted.
*1990-2010
**2000-2010

Source: EPA 2019a

COST-BENEFIT ANALYSIS IN THE REGULATORY PROCESS

If objectively and comprehensively conducted, a cost-benefit analysis should provide key information to regulatory decision-makers, elected policymakers, and the public. And while a full Regulatory Impact Analysis (RIA) should contain a variety of data and analyses, the cost-benefit analysis is a key conclusion.

The Office of Management and Budget'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" (OMB, 1-2).

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 the dollar-value of avoiding morbidity (illness) or premature mortality (shortened life span). The EPA has used diverse methodologies to monetize "workdays not lost" or "living longer," but the numbers have become so speculative and inflated as to have no meaningful predictive value.

This practice shields the EPA's rules with few measurable benefits from scrutiny. Furthermore, it subverts the purpose of cost-benefit analysis to compare the monetized cost and benefit of each regulation.

EPA's Study of Benefits and Costs of the Clean Air Act From 1990-2020

Most of the country already achieves the health-based National Ambient Air Quality Standards (NAAQS) for PM2.5. As of September 2019, only 18 out of 3,007 U.S. counties failed to meet the standard for PM2.5 (EPA 2019b). Under the federal Clean Air Act, the NAAQS for PM2.5 and the five other "criteria pollutants" must be set at a level protective of human health with an extra margin of safety, with no stipulation regarding cost of the regulations (United States Code). Thus, the NAAQS have been viewed as extremely conservative, precautionary standards. "It can also be argued that the 1970 Clean Air Act effectively operationalized the absolutist version of the precautionary principle" (Goklany, 4). Although variously defined, the precautionary principle generally means that with the risk of grave harm, however improbable 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. In risk assessments and analyses of the cost and benefits of regulation, it appears that the agency no longer regards the ambient pollutant levels set by the NAAQS to be fully protective. The Obama-era EPA attributed the risk of premature mortality at PM concentrations approaching and below natural (and thus unpreventable) background levels. Similarly, the EPA justified almost all of its new major air quality regulations on the basis of coincidental reduction of PM2.5 in rules not intended to address PM2.5 (see **Figure 3**).

The former EPA had an extraordinary focus on PM2.5—a criteria pollutant many scientists and regulators believe has already been reduced to healthy levels (Honeycutt, 32). To the past EPA, however, existing levels of PM2.5 pose risks to death on a par with cancer. A closer look at an EPA study issued in 2011 (EPA 2011a) reveals the questionable methodology and assumptions behind the EPA's preoccupation with ambient PM2.5. This study, *Benefits and Costs of the Clean Air Act: Second Prospective Study, 1990-2020* (referred to as the "Benefits study" in this paper), projects the benefits and the costs of the 1990 amendments to the CAA.

The EPA attributes 85 percent of the health benefits projected over the study period (1990-2020) to the reduction of ambient levels of PM2.5. This study finds that CAA regulation will "save" 230,000 lives by 2020 (EPA 2011a, 5-25). The EPA monetizes the value of those saved lives at nearly $2 trillion but estimates the annual direct compliance costs incurred at a comparatively paltry $65 billion (7-9).

The Obama EPA implied that the public pays only $1 for every $30 in health benefits as a result of additional reduction of ambient PM2.5. Over 90 percent of the $2 trillion derives from alleged prevention of "premature mortality"—roughly equivalent to shortened life expectancy. The EPA further imputed 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. "[T]he very wide margin between estimated benefits and costs, and the results of the uncertainty analysis, suggest that it is extremely unlikely that the monetized benefits of the CAAA [Clean Air Act Amendments] over the 1990 to 2020 period reasonably could be less than its costs, under any alternative set of assumptions we can conceive" (EPA 2011a, 7-8). The message was that more strict environmental regulations will, with nearly 100 percent certainty, provide a great return on investment.

If the claims about saving lives and gaining trillions of dollars in benefits were true, the case for an 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 much higher than from previous analyses of PM2.5 impacts and so lacking in credible explanation from the EPA that they exceed the bounds of credibility.

The numbers are so much higher than previous analyses of PM2.5 impacts and so lacking in credible explanation from the EPA that they exceed the bounds of credibility.

Peeling back the layers of assumptions 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. Tony Cox, Ph.D., 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" (Cox, 33). This is what in logic is called begging the question.

Assumption I: PM2.5 Causes Premature Mortality, aka Early Death

The main premise behind the EPA's promise of massive health benefits from additional regulation is that PM2.5 causes premature mortality or reduced lifespan. Yet, 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 PM2.5. The two studies on which the EPA relied for the Benefits study indicate statistical associations between mortality rates and PM2.5 concentrations in specific cities (Laden; Pope et al.). These chronic exposure studies exclude accidental death and adjust for other factors such as smoking or obesity but otherwise attribute all non-accidental deaths to PM2.5. 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 PM2.5 levels. *They cannot establish causation.* As an example, the statistical 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" (EPA 2011a, 5-40; emphasis added). After acknowledging this uncertainty, the EPA proceeds to the assumption that PM2.5 *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 (Bradford Hill). 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 PM2.5 and premature mortality. "Such complete certainty is unwarranted by available data and knowledge" (Cox, 820).

The EPA's attribution of the equivalent of 100 percent certainty to the assumption that PM2.5 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 and questioned whether the link between particulate matter and mortality is indeed causal or simply a result of model selection (Clyde). "Ecological epidemiology studies are not scientifically rigorous enough to draw conclusions about the cause of health effects identified in the studies for ozone or

any other pollutant and are not suitable for policy decisions" (Honeycutt, 27).

Many confounding variables left unaddressed in the EPA's selected studies weaken the credibility of the statistical association, and even more the assumption of a causal link between PM2.5 and premature mortality. Typical confounders include the presence of multiple pollutants commingled with PM2.5, the diverse composition of PM2.5—from natural dust to chemically enriched, and perhaps more hazardous, fine particles—and the question of whether earlier exposures to PM2.5 at levels far higher than current levels account for cumulative mortality risks later in life.

The question of exposure is a major confounder in many of the EPA's past 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 exposed to the monitored levels of outdoor PM2.5 is simply not a representative measure of actual exposure. Research shows that PM2.5 concentrations indoors are higher than outdoor levels. Yet cleaning the closet, vacuuming, cooking, or cruising through a department store can hardly be regarded as mortal risks (Valberg, 252-253).

PM2.5 concentrations indoors are higher than outdoor levels. Yet cleaning the closet, vacuuming, cooking, or cruising through a department store can hardly be regarded as mortal risks.

The EPA's past estimates of the benefits of reducing PM2.5-caused morbidity (sickness) also ignore key data to the contrary. The Benefits study projects 2.4 million fewer cases of aggravated asthma in 2020 due to PM2.5 regulations (EPA 2011a, 5-25). Yet the number of Americans with asthma has increased from 20 million to 25 million since 2001 (CDC 2018b), while concentrations of all CAA-regulated pollutants have declined by nearly 50 percent during that time (EPA 2019a).

The Obama EPA also disregarded studies that show no or even negative correlations. An analysis of mortality risks from PM2.5 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 (Thomas). Most importantly, the EPA ignored many toxicological and clinical studies, which are alone capable of evaluating whether, and to what extent, outdoor concentrations of PM2.5 may causally impact cardiopulmonary function.

Most toxicological studies contradict the past EPA's PM2.5 risk assessments. "Toxicologic 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 PM2.5 in the U.S. is not supported by the weight of scientific evidence, although other bases for regulating PM may be justifiable" (Green and Armstrong).

Assumption II: No Pollutant Threshold Below Which Air Is Healthy

In 2009, the EPA made a methodological change with huge ramifications. The agency began to calculate mortality risks from PM2.5 below the health-protective level of the NAAQS (presently set at an annual average of 12 $\mu g/m^3$). It also calculates benefits below the lowest measured ambient level (LML) in the original studies and even below natural background levels, all the way to zero. Remarkably, the EPA technical staff now assumes that there is no level of PM2.5 below which risks to premature death cease. Statisticians call this a "no threshold linear regression." 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 $\mu g/m^3$), the agency did not assume additional reductions in PM2.5 would generate additional health benefits. "However, starting in 2009, 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" (Smith, 23).

The statistical associations between premature mortality and PM2.5 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 PM2.5 levels for which no statistical evidence exists. "'Extrapolation' is the use of quantitative relationships outside the range of evidence on which it was based" (Smith, 23).

The EPA's adoption of this no-threshold approach increased the estimate of total U.S. deaths attributable to PM2.5 pollution from 88,000 to 320,000 (Smith, 24). This approach means, according to the EPA at least, that over two-thirds of the public's health risk from exposure to PM2.5 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 (24).

In short, the EPA's incredible finding is that mortal risks increase in proportion to the extent that a location's ambient concentration of PM2.5 exceeds natural background levels—now estimated by the EPA at the extremely low figure of 1 μg/m^3. "This created a major change in the level of national mortality estimated to be due to PM2.5 … because the majority of the US population resides in locations where the ambient PM2.5 concentrations are below 10μg/m^3" (Smith, 24).

Despite critical questions from members of Congress, senior EPA leadership defended adoption of the no-threshold approach. According to former Administrator Gina McCarthy, "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 at 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" (McCarthy, 1). This is another way of saying: No risk is too low, improbable, or uncertain that it is not worth regulating.

The Obama EPA claimed that the two studies in question show no evidence of a threshold, but many studies that they ignored do show a threshold. The agency's Benefits study admits that the "no-threshold" assumption is a "key uncertainty" but as usual assigns "high" confidence to the model that incorporates this assumption. The single study that EPA cited to support this questionable "no-threshold" assumption is one funded by the Health Effects Institute, which is in turn funded partly by the EPA (HEI).

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?

In spite of extremely low concentrations of PM2.5 in most areas of the country, the EPA did not 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.

A growing number of policymakers, state agencies, scientists, physicians, and concerned voters are baffled by the Obama-era EPA's inflated claims about low levels of PM2.5. Public disclosure of the data behind the EPA's claim has not been forthcoming even after repeated congressional requests and multiple subpoenas. Former U.S. Congressman Andy Harris, a medical doctor who chaired the Energy and Environment Subcommittee of the House Science, Space and Technology Committee, typified growing frustration with the lack of transparency in the EPA's science. "If our current air," he 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 that 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" (Harris, 6-7).

Public disclosure of the data behind the EPA's claim has not been forthcoming even after repeated congressional requests and multiple subpoenas.

Assumption III: Statistical Constructs Equals "Lives Saved"

Public pronouncements from the past EPA and from environmental advocates trumpet the dire need for additional regulation to save thousands of lives. Such unequivocal, emotional pronouncements grossly mislead the public. The EPA's Benefits study, for example, states that the authors "… estimate that cleaner air will, by 2020, prevent 230,000 cases of premature mortality in that year" (EPA 2011a). Former Administrator Jackson told the media that the public health protections in the Clean Air Act "… will mean the difference between sickness and health—in some cases, life and death—for hundreds of thousands of citizens" (Jackson 2011b).

Lives saved, deaths avoided, and premature mortality: the EPA's terms are misleadingly precise. "Avoided deaths" do not occur since clean air does not confer immortality, and these "saved lives" are nothing more than statistical constructs; they do not refer to real people. The health benefits the EPA projects from a regulatory reduction of PM2.5 is more accurately described as a reduction in the relative risk

Figure 2. Health benefits from PM2.5 reduction with alternative assumptions

	EPA Assumptions	Alternative Assumptions
Statistical lives	230,000	230,000
VSL/VSLY for median age of 80-year-old	$8.9 million	$1.5 million
Probability that assumption of true association: PM2.5 and premature death	100%	50%
Probability that association is causal	100%	50%
Probability of no PM2.5 threshold ambient health effects cease	100%	50%
Probability of reduction of health effects due to disease prevention and medication	N/A	50%
Totals	**$2.05 trillion**	**$21.6 billion**

VSL = Value of a Statistical Life
VSLY = Value of a Statistical Life Year

Source: Cox, 30-31

of mortality. Increased life-expectancy or life-years gained more accurately describe the health benefit at issue.

When not speaking for the public, the EPA calls these avoided deaths "statistical lives." The EPA constructs a "statistical life" (SL) by measuring the reduction in risks assumed to result from reduction of ambient PM2.5. "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" (Hildebrand, 18). Quite obviously, "statistical lives saved" bear no relationship to actual individual human lives.

For the thousands of lives that the EPA claimed air pollution has ended or that CAA regulation will save, not one single individual has been identified. Nor are there specific medical conditions or causes of death attributed to PM2.5 exposures. The past EPA's typical approach has been to assume any non-accidental death from cardiopulmonary conditions is caused by air quality. Yet, the CDC finds that smoking, not air pollution, accounts for 74 percent of deaths from chronic lower respiratory disease (CDC 2018a).

For the thousands of lives that the EPA claimed air pollution has ended or that CAA regulation will save, not one single individual has been identified.

This analysis also calls into question the $2 trillion monetary value of "preventing 230,000 deaths" in the Benefits study, which itself derives from a simple calculation. The EPA monetizes the value of one statistical life at $8.9 million at the estimated 2020 income level (EPA 2011a, 5-18). *Thus: 230,000 "prevented deaths" x $8.9 million per statistical life saved = $2.05 trillion.*

The valuation of one SL at $8.9 million is dubious. The past EPA estimated that the median age of people who gain additional life expectancy is 80 years (EPA 2011a, 5-28). And the increased life expectancy is estimated in several months, not years. When aggregated, however, into one statistical life, the EPA set a value of $8.9 million per statistical life-year gained. That figure is more appropriate for the monetized value of additional life expectancy for a 25 year old (Aldy 248) whereas the value for an 80 year old could be estimated at one-sixth of that value (Murphy and Topel, Figure 1). Thus, if a different but equally possible value for the octogenarian is used, the benefits decline by sixfold.

The EPA says there is insufficient evidence to adjust the base SL value for age (EPA 2011a, 5-22), but then they fail to note how sensitive their claims are to these assumptions that have very large uncertainties. The "EPA's evaluation of health benefits is unrealistically high, by a factor that could well exceed 1,000, and … it is therefore very likely that the costs of the 1990 CAAA exceed its benefits, plausibly by more than 50-fold" (Cox, 3).

Assumption IV: Co-Benefits of PM2.5 Reduction Can Justify Any Rule Under the CAA

The Obama-era EPA supported air quality regulations imposing multibillion-dollar costs on the basis of alleged

Figure 3. Degree of reliance on PM2.5-related co-benefits in RIAs

Year	RIAs for rules not targeting ambient PM2.5	PM co-benefits are >50% of total	PM co-benefits are only benefits quantified
2010	Reciprocating Internal Combustion Engines NESHAP -- Compression Ignition	✗	✗
2010	EPA/NHTSA Joint Light-Duty GHG & CAFES		
2010	SO_2 NAAQS (1-hr, 75 ppb)	✗	**> 99.9%**
2010	Mercury Cell Chlor Alkali Plant Mercury Emissions NESHAP	✗	
2011	Commercial & Industrial Solid Waste Incinerator Units NSPS	✗	✗
2011	Control of GHG from Medium & Heavy-Duty Vehicles		
2011	Utility Boiler MACT NSPS	✗	**> 99%**
2011	Sewage Sludge Incineration Units NSPS & Emission Guidelines	✗	✗
2012	Industrial, Commercial, and Institutional Boilers & Process Heaters NESHAP		
2012	Petroleum Refineries NSPS	✗	✗
2013	Existing Stationary Compression Ignition Engines NESHAP	✗	✗
2013	Existing Stationary Spark Ignition Engines NESHAP	✗	✗
2015	NSPS for GHGs from New Electric Utility Generating Units	✗	✗
2015	Clean Power Plan	✗	
2015	Ozone NAAQS, Final	✗	
2016	Oil and Gas Industry NSPS		
2016	Municipal Solid Waste Landfills NSPS		

Source: EPA 2019c

mortality risks from trace levels of PM2.5 created by the "no-threshold" approach. The EPA increasingly used these "coincidental reductions" of PM2.5 to justify the benefits of regulations intended to control not PM2.5 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 PM2.5 evidently started in 1997 when the EPA issued the first NAAQS for PM2.5. Since 2009, however, the EPA has increasingly used PM2.5 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 SO_2 NAAQS rely on co-benefits from PM2.5 reduction for over 99 percent of estimated health benefits (EPA 2019c). Without these co-benefits, the EPA's estimate of the direct costs of these rules would far exceed any measurable benefits.

With the mercury rule, the EPA admitted that the direct health benefits from reduction of mercury accounted for only 0.007 percent (or $6 million) of the $90 billion in total health benefits. Reductions in CO_2 emissions added another 0.4 percent ($360 million), while PM2.5 co-benefits accounting for the remaining 99.593 percent (Federal Register 77, 9306). The EPA estimated the direct costs of the rule at $9.6 billion. The agency's press releases and congressional testimony do not acknowledge this huge gap between direct mercury benefits and indirect PM2.5 benefits, but the Federal Register notice for this rule explicitly reveals the glaring gap (9306).

As shown in **Figure 3**, in 12 RIAs for rules not targeting PM2.5, submitted between 2010 and 2016, co-benefits from PM2.5 accounted for more than half of all estimated health benefits. In nine of the cost-benefit analyses, co-benefits from PM2.5 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 PM2.5 in regulations not targeting this pollutant. By relying on co-benefits from PM2.5, the EPA also evades its obligation to justify the need for stricter regulations. Consider again 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. Roughly 95 percent of the 11,000 (statistical) lives "saved" by the mercury rule derived from PM2.5 co-benefits in geographical areas that already attained the PM2.5 NAAQS of 12 μg/m^3 (EPA 2011b, 5-102).

Recall that the NAAQS are conservative federal standards below which human health should be 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 PM2.5 is one. If the EPA is convinced that ambient PM2.5 now presents dire health risks, the agency should make its case for strengthening the PM2.5 NAAQS. The EPA lowered the 15 μg/m^3 national standard for PM2.5 to 12 μg/m^3, and the current 12 μg/m^3 standard is again under review.

As Anne E. Smith, Ph.D., of National Economic Research Associates (NERA) noted in a thoroughly researched analysis of the EPA's use of co-benefits, "EPA's PM2.5 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 … This 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 the increased regulatory burden that new rulemakings create" (Smith, 15).

Her conclusion is equally on point. "In all, EPA's use of co-benefits in its RIAs 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" (Smith, 33).

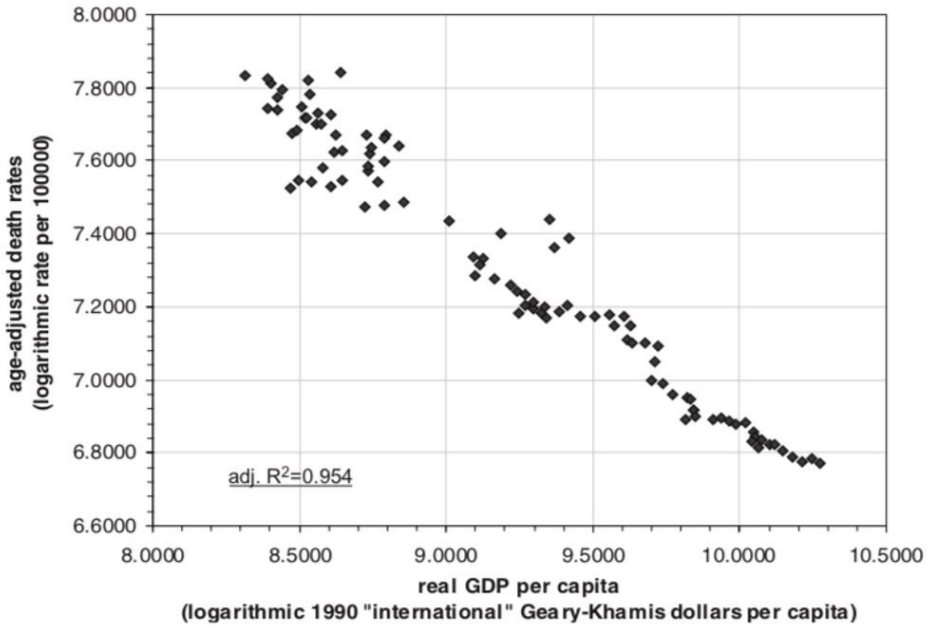
Recommendations

The Obama-era EPA environmental science had a distinct pattern that needs to be revised by the current EPA. The agency relied on one or two cherry-picked studies which indicated the most adverse health effects at the lowest concentration of the pollutant in question. The EPA either ignored or gave lip service to sometimes hundreds of equally reputable studies that contradict the studies presented by the EPA. The EPA's favored studies have usually been 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 declared complete certainty and approval by peer review.

Sound science and objective scientists abound. Public health science in the hands of government, however, is easily compromised in order to reach predetermined policy outcomes. The Obama EPA would have the public believe that "pure science" shows that a regulatory agenda to supplant fossil fuels is necessary to save the lives of hundreds of thousands. If their policy objective was to supplant fossil fuels, PM2.5 is a useful tool. PM2.5 is an ever-present byproduct of combustion of coal, natural gas, and oil. Emissions from new cars and trucks, however, are 98-99 percent lower compared to the 1960s, reducing many pollutant levels over 90 percent as vehicle miles traveled more than doubled (EPA 2018a). On the other hand, natural processes will always release fine particles into the ambient atmosphere of this planet.

Environmental regulatory standards reflect a judgment about what is acceptable or unacceptable societal risk. As such, the EPA's final regulatory decisions

Figure 4: Health effects of poverty and unemployment



Source: Brenner

are informed by science but ultimately are policy decisions that no scientific findings can dictate in our democracy. 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, consistent with the data in **Figure 4**.

The Clean Air Act under which the EPA conducts risk assessment and sets national standards should stipulate minimal criteria for scientific risk assessment of health effects sufficiently robust to guide decisions on air quality standards. Such minimal criteria could include the following:

- The EPA's risk assessments must be peer-reviewed by an independent body.
- 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.
- Abandon no-threshold modeling assumptions in setting ambient standards and regulatory emission limits.
- Health-based air quality standards should 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.
- Use a plausible biological mechanism as predicate for health-effects findings.
- Conduct a comprehensive, cumulative cost-benefit analysis of all rules according to methodology and scope stipulated in law.

Conclusion

The EPA's regulatory sway is at a tipping point as the Trump administration works to restore sound cost-benefit analyses based on objective principles, instead of manipulating such analyses to justify the most stringent regulations possible. Updating the estimates for the value of a statistical life (EPA 2018b) and making a clear distinction between science and policy considerations during the NAAQS review process (Pruitt, 10) are two important reforms being undertaken. However, a future administration can easily undo these reforms, which is why Congress should reform the Clean Air Act and ensure the environmental regulatory process is not subject to the whims of the current president and the administrative state.

EPA's Pretense of Science: Regulating Phantom Risks *was initially released in 2012. This updated version uses the most recent data available.*

References

Aldy, Joseph E. and W.K. Viscusi. 2007. "Age Differences in the Value of a Statistical Life: Revealed Preference Evidence." *Review of Environmental Economics and Policy* 1(2): 241-260.

Bradford-Hill, Austin. 1965. "The Environment and Disease: Association or Causality?" *Proceedings of the Royal Society of Medicine* 58(5): 295–300.

Brenner, M. Harvey. 2005. "Commentary: economic growth is the basis of mortality rate decline in the 20th century—experience of the United States 1901-2000." *International Journal of Epidemiology* 34(6): 1214-1221.

CDC (Centers for Disease Control and Prevention). 2017a. "FastStats – Cancer." Last updated May 3. Accessed July 23, 2019.

CDC (Centers for Disease Control and Prevention). 2017b. "FastStats – Deaths and Mortality." Last updated May 3. Accessed October 14, 2019.

CDC (Centers for Disease Control and Prevention). 2018a. "Tobacco-Related Mortality." Last updated January 17. Accessed October 28, 2019.

CDC (Centers for Disease Control and Prevention). 2018b. "National Health Interview Survey Asthma Prevalence Data." Last updated April 18. Accessed September 30, 2019.

CFR (Code of Federal Regulations). 2013a. "National primary ambient air quality standards for $PM_{2.5}$." Title 40. Section 50.18.Clyde, Merlise. 2000. "Model Uncertainty and Health Effects Studies for Particulate Matter." *Environmetrics* 11(6): 745-763.

Cox, Louis Anthony (Tony), Jr. 2012. "Reassessing the Human Health Benefits of Cleaner Air." *Risk Analysis* 32(5): 816-829.

EPA (Environmental Protection Agency). 2011a. *The Benefits and Costs of the Clean Air Act from 1990-2020.* EPA.

EPA (Environmental Protection Agency). 2011b. *Regulatory Impact Analysis for the Final Mercury and Air Toxics Standards*. EPA.

EPA (Environmental Protection Agency). 2015. *Regulatory Impact Analysis for the Clean Power Plan Final Rule*. EPA.

EPA (Environmental Protection Agency). 2018a. "History of Reducing Air Pollution from Transportation in the United States." Last updated April 19.

EPA (Environmental Protection Agency). 2018b. "Mortality Risk Valuation: What is the current process for updating the Agency's estimates?" Last updated February 8.

EPA (Environmental Protection Agency). 2019a. "Air Quality Trends – National Summary." Last updated July 8.

EPA (Environmental Protection Agency). 2019b. "PM-2.5 (2012) Nonattainment Area Summary." Last updated August 31.

EPA (Environmental Protection Agency). 2019c. "Regulatory Impact Analyses for Air Pollution Regulations." Last updated August 29.

Federal Register. 2012. Volume 77, No. 32, 9306. February 16.

Green, Laura C., and Sarah R. Armstrong. 2003. "Particulate Matter in Ambient Air and Mortality: Toxicologic Perspectives." *Regulatory Toxicology and Pharmacology* 38(3): 326-335.

Goklany, Indur M. 2001. "The Precautionary Principle: A Critical Appraisal of Environmental Risk Assessment." *Cato Institute*.

Harris, Andy. 2013. "Opening Statement before the U. S. House of Representatives Subcommittee on Energy and the Environment, Committee on Science, Space and Technology." Committee on Science, Space, and Technology.

HEI (Health Effects Institute). 2019. "What is the Health Effects Institute?" Accessed October 21.

Hildebrand, Susan, Michael Honeycutt, and Stephanie Shirley. 2012. *EPA's Cost Benefit Analysis*. Texas Commission on Environmental Quality.

Honeycutt, Michael. 2011. *Hearing on Quality Science for Quality Air*. Testimony to the U.S. House Committee on Space, Science, and Technology, Subcommittee on Energy and Environment. October 4.

Jackson, Lisa P. 2011a. *Hearing on Regulatory Reform Series #7: The EPA's Regulatory Planning, Analysis, and Major Actions*. Testimony to the U.S. House Committee on Energy and Commerce, Subcommittee on Oversight and Investigations, September 22.

Jackson, Lisa P. 2011b. "Too Dirty to Fail?" *Los Angeles Times,* October 21.

Koop, Gary, and Lise Tole. 2004. "Measuring Health Effects of Air Pollution: To What Extent Can We Really Say People Are Dying from Bad Air?" *Journal of Environmental Economics and Management.* 47(1): 30-54.

Koop, Gary, and Lise Tole. 2006. "An Investigation of Thresholds in Air Pollution—Mortality Effects." *Environmental Modeling & Software* 21(12): 1662-1673.

Laden, Francine. Joel Schwartz, Frank E. Speizer, and Douglas W. Dockery. 2006. "Reduction in Fine Particulate Air Pollution and Mortality." *American Journal of Respiratory and Critical Care Medicine* 173: 667-672.

McCarthy, Gina. 2012. "Letter to Rep. Fred Upton." Environmental Protection Agency, February 3.

Milloy, Steve. 2012. "Clearing the Air on the EPA." *Washington Times*, March 7.

Murphy Kevin, and Robert Topel. 2003. "The Economic Value of Medical Research." In *Measuring the Gains from Economic Research: An Economic Approach*, edited by Kevin M. Murphy and Robert H. Topel, 9-40. Chicago: University of Chicago Press.

OMB (Office of Management and Budget). 2003. "Circular A-4." September 17.

Pope III, C. Arden, Richard T. Burnett, Michael J. Thun, Eugenia E. Calle, Daniel Krewski, Kazuhiko Ito, and George D. Thurston. 2002. "Lung Cancer, Cardiopulmonary Mortality, and Long-Term Exposure to Fine Particulate Air Pollution." *Journal of the American Medical Association* 287(9): 1132-1141.

Pruitt, E. Scott. 2018. "Back-to-Basics Process for Reviewing National Ambient Air Quality Standards." Environmental Protection Agency, May 9.

Reis, Patrick, Erica Martinson, Darius Dixon, and Darren Samuelsohn. 2011. "House to Vote on Carbon Cap - Oil Majors to Announce Q3 Profits - *Reason Magazine vs. Grist vs. the Media* - Supercommittee Courted by Superlobby." *Politico*, October 24.

Ropeik, David, and George Gray. 2002. *Risk: A Practical Guide for Deciding What's Really Safe and What's Really Dangerous in the World Around You.* Boston: Houghton Mifflin.

Smith, Anne E. 2011. "An Evaluation of the PM2.5 Health Benefits Estimates in Regulatory Impact Analyses for Recent Air Regulations." *NERA Economic Consulting*, December 31.

Thomas, Duncan C., Michael Jerrett, Nino Kuenzli, Thomas A. Louis, Francesca Dominici, Scott Zeger, Joel Schwarz, Richard T. Burnett, Daniel Krewski, and David Bates. 2007. "Bayesian Model Averaging in Time-Series Studies of Air Pollution and Mortality." *Journal of Toxicology and Environmental Health Part A* 70(3-4): 311-315.

United States Code. Title 42, Chapter 85, Section 7409.

Valberg, Peter A. 2011. "Comments on U.S. EPA's Projections of Mortality Reductions Achieved by Reducing Levels of Particulate in Our Ambient (Outdoor) Air." Testimony to the U.S. House Committee on Energy and Commerce, Subcommittee on Energy and Power, September 8.

White, Kathleen H. 2012. *EPA's Approaching Regulatory Avalanche.* Texas Public Policy Foundation.

WJXT (Jacksonville Television). 2012. "EPA administrator fires back on GOP attacks." News4Jax, January 6.

WSJ (*Wall Street Journal*) Editorial Board. 2011. "Boiler Room Politics." *Wall Street Journal*, March 4.

ABOUT THE AUTHORS

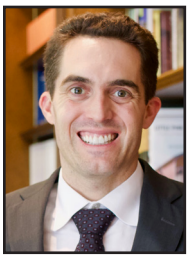
The Honorable Kathleen Hartnett White joined the Texas Public Policy Foundation in January 2008. She is the director of the Armstrong Center for Energy & the Environment, a senior fellow for the Life:Powered project, and a distinguished senior fellow-in-residence.

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 and utilities, storage and disposal of waste, TCEQ's staff of 3,000, annual budget of over $600 million, and 16 regional offices make it the second largest environmental regulatory agency in the world after the U.S. Environmental Protection Agency.

In 2016 White was named to the Trump Campaign Economic Advisory Council. She was twice nominated by President Trump to chair the White House Council of Environmental Quality and has served on more than 10 national and state commissions to include law under a Lineberry Foundation Fellowship at the Law School of Texas Tech University.

Prior to Gov. Rick Perry's appointment of White to the TCEQ in 2001, she served as then-Governor George Bush's appointee to the Texas Water Development Board where she sat until appointed to TCEQ. She served on the Texas Economic Development Commission and the Environmental Flows Study Commission and also completed her term as an officer and director of the Lower Colorado River Authority. In 2016, Regnery Publishing released her book *Fueling Freedom: Exposing the Mad War on Energy* co-authored with Stephen Moore. Her writing has appeared in numerous publications including *National Review, Investors' Business Daily, Washington Examiner, Forbes, Daily Caller, The Hill*, and major Texas newspapers. She regularly testifies before the U.S. Congress, including the U.S. Senate Environment and Public Works Committee and the House Committee on Science.

A writer and consultant on free market natural resource policy, private property rights, and ranching history, White received her bachelor's *cum laude* and master's 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 and her husband are partners of a fifth generation family ranch in Presidio, Texas, and are members of the Texas Hereford Association and the American Hereford Association. She is a former commissioner of the Texas Strategic Economic Development Planning Commission and a former board member of the Texas Wildlife Association and the National Cattlemen's Legal Defense Fund.



Brent Bennett, Ph.D., is a policy analyst for Life:Powered, a project of the Texas Public Policy Foundation, which seeks to raise America's energy IQ and promote energy policies that advance freedom and human flourishing around the world. Prior to joining Life:Powered, Dr. Bennett worked as an engineer for a battery materials startup, and he continues to provide technology consulting to battery companies. He was raised in Midland, Texas, and was inspired to study energy because of his upbringing in the heart of the oil and gas industry. He has a B.S. in physics from the University of Tulsa and an M.S.E. and Ph.D. in materials science and engineering from the University of Texas at Austin. His graduate research focused on new chemistries for utility-scale energy storage systems, and he complemented his oil and gas background with in-depth knowledge of renewable energy technologies and utility markets.

About Texas Public Policy Foundation

The Texas Public Policy Foundation is a 501(c)3 non-profit, non-partisan research institute. The Foundation promotes and defends liberty, personal responsibility, and free enterprise in Texas and the nation by educating and affecting policymakers and the Texas public policy debate with academically sound research and outreach.

Funded by thousands of individuals, foundations, and corporations, the Foundation does not accept government funds or contributions to influence the outcomes of its research.

The public is demanding a different direction for their government, and the Texas Public Policy Foundation is providing the ideas that enable policymakers to chart that new course.

Texas Public Policy Foundation

901 Congress Avenue | Austin, Texas 78701 | 512.472.2700 | www.TexasPolicy.com