WRITTEN REPORT OF GEORGE D. THURSTON
REGARDING THE PUBLIC HEALTH BENEFITS OF EPA'S PROPOSED RULEMAKING REGARDING BEST AVAILABLE RETROFIT TECHNOLOGY FOR TEXAS SOURCES UNDER THE REGIONAL HAZE RULE

RE: ENVIRONMENTAL PROTECTION AGENCY,
Promulgation of Air Quality Implementation Plans; State of Texas; Regional Haze and Interstate Transport of Pollution Affecting Visibility Federal Implemntation Plan, 82 Fed. Reg. 912 (proposed Jan. 4, 2017)
EPA Docket No.: EPA–R06–OAR–2016–0611; FRL–9955–77-Region 6

May 4, 2017

PROFESSIONAL EXPERIENCE OF THE AUTHOR

I am Professor of Environmental Medicine at the New York University (NYU) School of Medicine.

I have a Bachelor of Science degree in Engineering from Brown University, and a Masters and Doctorate of Environmental Health Sciences from the Harvard University School of Public Health. I have over 30 years of subsequent experience in the evaluation of the human health effects of air pollution. I have served on the U.S. Environmental Protection Agency's Clean Air Scientific Committee (CASAC) that advises the EPA on the promulgation of ambient air quality standards from 2007 through 2010, and I have served on the National Academy of Science's Committee on the Health Effects of Incineration from 1995 through 1999. I have published extensively regarding the health effects of inhaled air pollutants on humans, particularly as it relates to asthma attacks, hospital admissions, and mortality, in prominent scientific journals, such as Science, Lancet, Thorax, and The Journal of the American Medical Association (JAMA). I have also been called upon by both the U.S. House of Representatives and the U.S. Senate on multiple occasions in recent decades to provide testimony before them regarding the human health effects of air pollution, most recently on October 10, 2010. A statement of my qualifications is attached to my affidavit as Exhibit T-1.

SUMMARY OF REPORT

The purpose of this report is to document the adverse human health effects that are associated with exposures to air pollutants from fossil fuel-fired utility power plants generally, and in particular, the adverse human health effects that will be avoided by the application of EPA's proposed sulfur dioxide (SO_2) emission limits for 18 individual electric generating units (EGU) at nine power plants in Texas.

This report documents how emissions from these 18 EGUs contribute to the serious and well-documented adverse human health effects known to be associated with exposure to air pollution from fossil fuel-fired power plants. The documentation I present confirms this conclusion, including both epidemiological and toxicological evidence that I and others have published in the medical and scientific literature. In this work, I also rely upon the expert report submitted by Dr. Gray. Applying this information to the U.S. EPA approved Environmental

Benefits Mapping and Analysis Program (BenMAP) model, I then provide calculations of the excess adverse human health impacts that would occur each year if EPA's proposed BART controls for these 18 Texas EGUs are not installed, as well as the annual economic valuation of those health impacts across 14 states.[1]

BACKGROUND

The adverse health consequences of breathing air pollution from sources such as fossil-fuel fired utility power plants are well documented in the published medical and scientific literature. During the past decades, medical research examining air pollution and public health has shown that air pollution is associated with a host of serious adverse human health effects. This documentation includes impacts revealed by observational epidemiology, and confirmed by controlled chamber exposures, showing consistent associations between air pollution and adverse impacts across a wide range of human health outcomes.

Observational epidemiology studies provide the most compelling and consistent evidence of the adverse effects of air pollution. "Epidemiology" is literally "the study of epidemics," but includes all statistical investigations of human health and potentially causal factors of good or ill health. In the case of air pollution, such studies follow people as they undergo varying real-life exposures to pollution over time, or from one place to another, and then statistically inter-compare the health impacts that occur in these populations when higher (versus lower) exposures to pollution are experienced. In such studies, risks are often reported in terms of a Relative Risk (RR) of illness, wherein a RR =1.0 is an indication of no change in risk after exposure, while a RR>1.0 indicates an increase in health problems after pollution exposure, and that air pollution is damaging to health.

These epidemiological investigations are of two types: 1) population-based studies, in which an entire city's population might be considered in the analysis; and 2) cohort studies, in which selected individuals, such as a group of asthmatics, are considered. Both of these types of

[1] In April 2015, I prepared a separate report documenting the human health benefits across ten states resulting from EPA's proposed sulfur dioxide emission reductions at 14 Texas EGUs. *See* U.S. Environmental Protection Agency, Approval and Promulgation of Implementation Plans; Texas and Oklahoma; Regional Haze State Implementation Plans; Interstate Transport State Implementation Plan To Address Pollution Affecting Visibility and Regional Haze; Federal Implementation Plan for Regional Haze and Interstate Transport of Pollution Affecting Visibility; Proposed Rule, 79 Fed. Reg. 74,818 (Dec. 16, 2014), EPA Docket No. EPA-R06-OAR-2014-0754-0070.

epidemiologic studies have shown confirmatory associations between air pollution exposures and increasing numbers of adverse impacts, including:

- decreased lung function (a measure of our ability to breathe freely);
- more frequent asthma symptoms;
- increased numbers of asthma and heart attacks;
- more frequent emergency department visits;
- additional hospital admissions; and
- increased numbers of deaths.

The fact that the effects of air pollution have been shown so consistently for so many health endpoints, and in so many locales, indicates these associations to be causal.

Fine Particulate Matter (PM) is among the key air pollutants emitted from power plants that have been revealed by research to adversely affect human health. These research studies have been conducted for a wide array of geographic areas, including eastern North America. $PM_{2.5}$ air pollution has been carefully studied in recent decades. PM is composed of two major components: "primary" particles, or soot, emitted directly into the atmosphere by pollution sources, and; "secondary" particulate matter, formed in the atmosphere from gaseous pollutants, such as the sulfur oxides (SOx) and nitrogen oxides (NOx) also emitted by coal-fired power plants. After formation in the atmosphere, this secondary PM largely condenses upon the smallest existing primary particles that, collectively, represent the greatest surface area for the secondary PM to condense upon. These particles are very small, commonly having an aerodynamic diameter of less that 1.0 micrometer (*u*m) – a fraction of the diameter of a human hair. For example, after it is released from a smokestack, gaseous SOx is chemically converted in the atmosphere to become sulfate PM.

In addition to lung damage, recent epidemiological and toxicological studies of PM air pollution have shown adverse effects on the heart, including an increased risk of heart attacks. For example, when PM stresses the lung (*e.g.*, by inducing edema), it places extra burden on the heart, which can induce fatal complications for persons with cardiac problems. Indeed, for example, Peters et al. (2001) found that elevated concentrations of fine particles in the air can elevate the risk of Myocardial Infarctions (MI's) within a few hours, and extending 1 day after PM exposure. The Harvard University team found that a 48 percent increase in the risk of MI was associated with an increase of 25 $u g/m^3$ $PM_{2.5}$ during a 2-hour period before the onset of MI,

and a 69 percent increase in risk to be related to an increase of 20 *u*g/m^3 $PM_{2.5}$ in the 24-hour average 1 day before the MI onset (Peters et al., 2001). Numerous other U.S. studies have also shown qualitatively consistent acute cardiac effects, such as the Sullivan et al. (2005) study of acute myocardial infarctions in King County, Washington; the Zanobetti and Schwartz (2006) study of hospital admissions through emergency departments for myocardial infarction (ICD-9 code 410); and the Zanobetti et al. (2009) study that examined the relationship between daily $PM_{2.5}$ concentrations and emergency hospital admissions for cardiovascular causes, myocardial infarction, and congestive heart failure in 26 U.S. communities during 2000-2003.

Cardiac effects at the biological level have also been documented in both animal and human studies. Animal experiments at Harvard University by Godleski et al. (1996, 2000) indicate that exposures to elevated concentrations of ambient PM can result in cardiac related problems in dogs that had been pre-treated (in order to try to simulate sensitive individuals) to induce coronary occlusion (i.e., narrowed arteries in the heart) before exposing them to air pollution. The most biologically and clinically significant finding was that, in these dogs, the PM affected one of the major electrocardiogram (ECG) markers of heart attacks (myocardial ischemia) in humans, known as elevation of the ST segment. Cardiac effects at the biological level have been found in human studies, as well. For example, Pope et al. (1999) and Gold et al. (1999) found that PM exposure is associated with changes in human heart rate variability. Such changes in heart rate variability (HRV) may reflect changes in cardiac autonomic function and risk of sudden cardiac death. In the Pope et al. study, repeated ambulatory ECG monitoring was conducted on 7 subjects for a total of 29 person-days before, during, and after episodes of elevated pollution. After controlling for differences across patients, elevated particulate levels were found to be associated with (1) increased mean heart rate, (2) decreased SDNN, a measure of overall HRV, (3) decreased SDANN, a measure that corresponds to ultra-low frequency variability, and (4) increased r-MSSD, a measure that corresponds to high-frequency variability. This confirms, at the individual level, that biological changes do occur in heart function as a result of PM exposure, supporting the biological plausibility of the epidemiological associations between PM exposure and cardiac illnesses.

Epidemiologic research conducted on U.S. residents has indicated that acute exposure to PM air pollution is associated with increased risk of mortality. A nationwide time-series statistical analysis by the Health Effects Institute (HEI, 2003) of mortality and PM_{10} air pollution in 90 cities across the US indicates that, for each increase of 10 *u*g/m^3 in daily PM_{10} air pollution

concentration, there is an associated increase of approximately 0.3% in the *daily* risk of death. While a 0.3 % change in the daily death risk may seem small, it is important to realize that such added risks apply to the entire population, and accumulate day after day, week after week, and year after year, until they account for thousands of needless daily deaths from air pollution in the U.S. each year. Indeed, I concur with the most recent U.S. EPA Particulate Matter Integrated Science Assessment (ISA) (USEPA, 2009), which unequivocally states that "Together, the collective evidence from epidemiologic, controlled human exposure, and toxicological studies is sufficient to conclude that *a causal relationship exists between short term exposures to $PM_{2.5}$ and cardiovascular effects . . . and mortality.*"[2]

In addition to the acute health effects associated with daily PM pollution, the long-term exposure to fine PM is also associated with increased lifetime risk of death and has been estimated to take years from the life expectancy of people living in the most polluted cities, relative to those living in cleaner cities. For example, in the Six-Cities Study (which was one key basis for the setting of the original $PM_{2.5}$ annual standard in 1997), Dockery et al. (1993) analyzed survival probabilities among 8,111 adults living in six cities in the central and eastern portions of the United States during the 1970's and 80's. The cities were: Portage, WI (P); Topeka, KS (T); a section of St. Louis, MO (L); Steubenville, OH (S); Watertown, MA (M); and Kingston-Harriman, TN (K). Air quality was averaged over the period of study in order to study long-term (chronic) effects. As shown in Figure 1, it was found that the long-term risk of death, relative to the cleanest city, increased with fine particle exposure, even after correcting for potentially confounding factors such as age, sex, race, smoking, etc.

In addition, a study that I wrote with co-authors, published in the Journal of the American Medical Association (JAMA), shows that long-term exposure to combustion-related fine particulate air pollution is an important environmental risk factor for cardiopulmonary and lung cancer mortality. Indeed, as shown in Figure 2, this study indicates that the increase in risk of lung cancer from long-term exposure to $PM_{2.5}$ in a city like New York was of roughly the same size as the increase in lung cancer risk of a non-smoker who breathes passive smoke while living with a smoker, or about a 20% increase in lung cancer risk. *See* Pope, CA, et al., 2002.

[2] U.S. Environmental Protection Agency (2009a) (emphasis added).

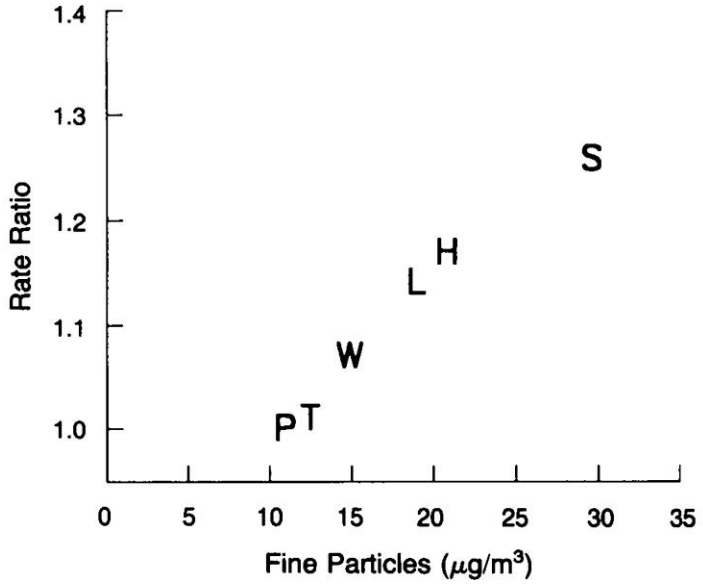

Figure 1. The Harvard Six-Cities Study showed that the lifetime risk of death increased across 6 U.S. cities as the average fine PM levels increased. (Source: Dockery et al., 1993).

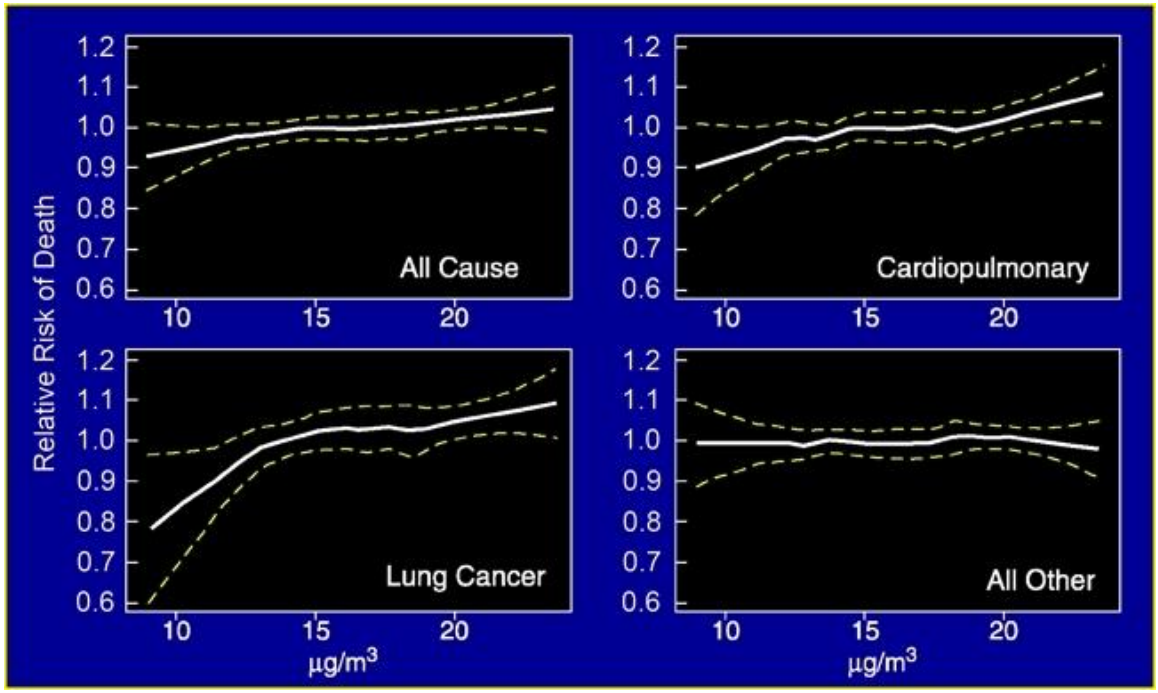

Figure 2. Cardiopulmonary and lung cancer mortality risks increase monotonically with exposure to long-term fine PM (adapted from: Pope, Burnett, Thun, Calle, Krewski, Ito, and Thurston, 2002)

Most studies evaluate whether rising air pollution levels worsen health, but it has also been shown that reducing pollution in the air can result in health benefits to the public. For

example, Pope (1989) conducted a compelling study clearly showing that, when pollution levels diminish, the health of the general public improves. He investigated a period during the winter of 1986-87 when the Geneva Steel mill in the Utah Valley shut down during a strike. The PM levels dropped dramatically in that strike-year winter, as opposed to the winters preceding and following when the steel mill was in operation. As shown in Figure 3 below, hospital admissions in the valley showed the same pattern as the PM air pollution, decreasing dramatically during the strike. As a control, Pope also examined the pollution and hospital admissions records in nearby Cache Valley, where the mill's pollution was not a factor, and no such drop in respiratory admissions was seen, showing that the drop in admissions in the Utah Valley was not due to some cause other than the reduction in the air pollution levels.

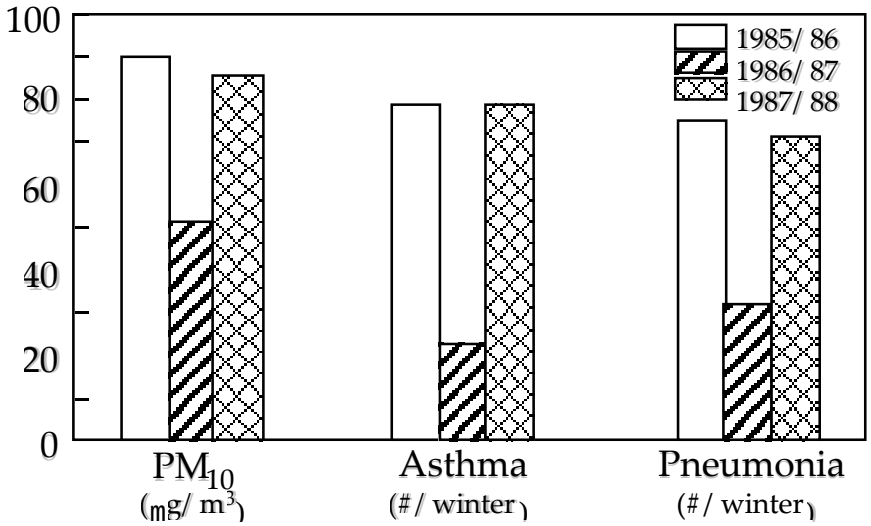

Figure 3. Decreasing PM pollution lowered the number of children's hospital admissions (Source: Pope, 1989).

These studies of the health improvements associated with decreases in $PM_{2.5}$ pollution show that any reduction can be expected to result in commensurate health benefits to the public at ambient levels, even where the National Ambient Air Quality Standards (NAAQS) are already met. A follow-up analysis of the Harvard Six-Cities Study cohort discussed earlier (Dockery et al., 1993), published in the March 15, 2006 issue of The American Journal of Respiratory and Critical Care Medicine (Laden et al., 2006), shows that mortality is decreased by lowering PM pollution. This study was carried out in the same six metropolitan areas evaluated in the earlier study, study participants' ages ranged from 25 to 74 at enrollment in 1974, and the scientists tracked both PM air pollution and mortality through 1998 in these populations. The Laden study

found that improved overall mortality (i.e., a risk ratio significantly below 1.0) was associated with decreased mean $PM_{2.5}$ over the study follow-up time (RR = 0.73; 95% per 10 $\mu g/m^3$, CI = 0.57-0.95). In other words, for each decrease of 1 $\mu g/m^3$ of $PM_{2.5}$, the overall death rate from causes such as cardiovascular disease, respiratory illness and lung cancer decreased by nearly 3% (i.e., 10 $\mu g/m^3$ x 2.7% = 27% decrease, or RR=0.73). The study also found that people who are exposed to lower pollution live longer than they would if they were exposed to higher pollution. Francine Laden, the study's lead author, explained its key findings in the March 21, 2006 issue of the New York Times: "For the most part, pollution levels are lower in this country than they were in the 70's and 80's," and "the message here is that if you continue to decrease them, you will save more lives."[3] "Consistently," Dr. Laden said, "in the cities where there was the most cleanup, there was also the greatest decrease in risk of death."

Although the Laden study took place in urbanized areas, the same principle can be applied in more rural areas where the air is more pristine: higher concentrations of $PM_{2.5}$, even at very low overall levels, are associated with greater health risks. Indeed, a more recent Canadian national-level cohort study, Crouse et al. (2012), has shown that the adverse effects of air pollution extend down to very low levels of $PM_{2.5}$. These investigators calculated hazard ratios (i.e., risk ratios) and 95% confidence intervals (CIs), adjusted for available individual-level and contextual covariates, finding a relative risk (or hazard ratio) of 1.30 (95% CI: 1.18, 1.43) for cardiovascular mortality from Cox proportional hazards survival models with spatial random-effects. Figure 4, taken from the Crouse study, illustrates the finding that mortality risk decreases with decreasing levels of $PM_{2.5}$, even at ambient $PM_{2.5}$ levels down to 1 $\mu g/m^3$.

[3] Nicholas Bakalar, *Cleaner Air Brings Drop in Death Rate*, New York Times (Mar. 21, 2006), pg F7.

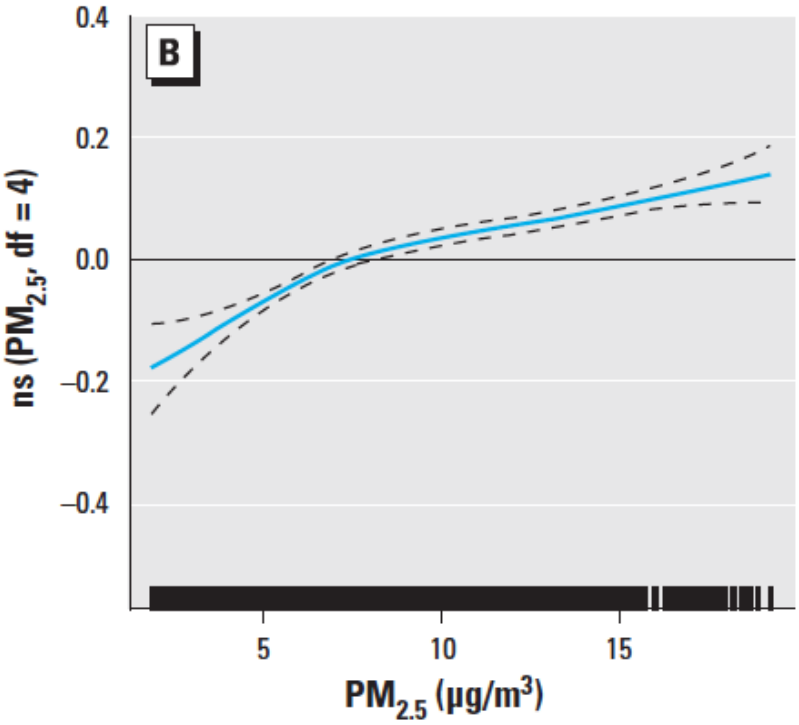

Figure 4. Cardiovascular Mortality Risk vs. $PM_{2.5}$ exposure (solid line) and 95% CIs (dashed lines), showing increasing risk of death with increasing $PM_{2.5}$, even at very low ambient levels of $PM_{2.5}$ air pollution (from Crouse et al., 2012).

Similarly, my own research has verified (as shown in Figure 5) that the association between $PM_{2.5}$ air pollution and cardiovascular mortality extends down to very low $PM_{2.5}$ concentration levels in the US as well (Thurston et al, 2016). Importantly, this study is highly regarded, as it was conducted in a well characterized and large US population: the National Institutes of Health – American Assoiation of Retired Persons (NIH-AARP) Diet and Health Study cohort. The NIH-AARP Study was initiated when members of the AARP, aged 50 to 71 years from 6 US states (California, Florida, Louisiana, New Jersey, North Carolina, and Pennsylvania) and 2 metropolitan areas (Atlanta, Georgia, and Detroit, Michigan), responded to a mailed questionnaire in 1995 and 1996. The NIH-AARP cohort questionnaires elicited information on demographic and anthropometric characteristics, dietary intake, and numerous health-related variables (e.g., marital status, body mass index, education, race, smoking status, physical activity, and alcohol consumption), that was used to control for these factors in the air pollution mortality impact assessment.

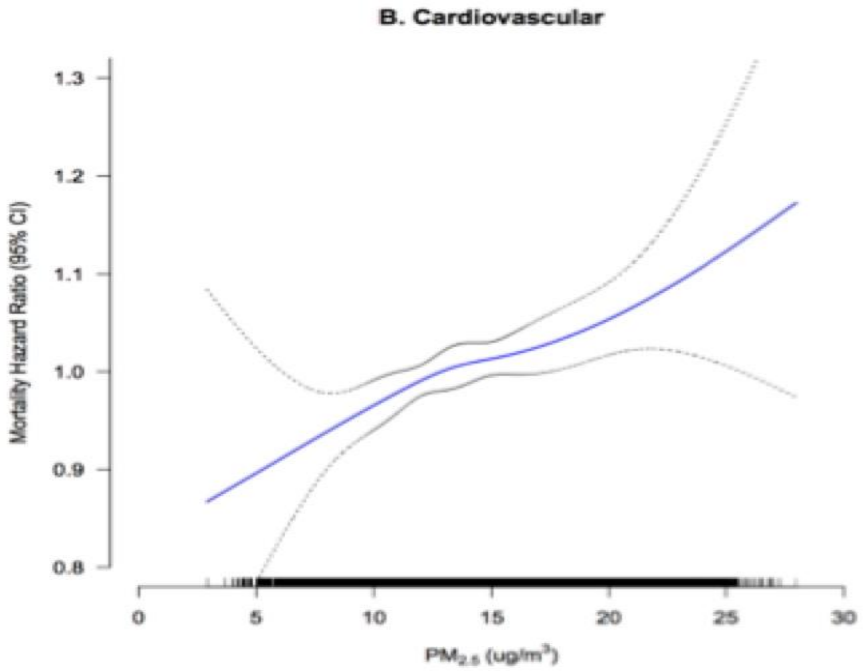

Figure 5. Mortality Risk from Cardiovascular Disease Increases with Rising PM2.5 Exposure, Even Well Below the Present US Ambient Air Quality Standard annual limit for PM2.5 (12 µg/m3). Thurston *et al*., 2016a.

Although published too late to be considered by the U.S. EPA in their 2013 standard setting process, the Crouse et al. (2012) and Thurston et al. (2016a) results indicate that the mortality effects of $PM_{2.5}$ air pollution can occur at even lower ambient air pollution levels than shown by Pope et al. 2002, and even lower levels than that at which the U.S. EPA assumed the effects of $PM_{2.5}$ to exist in its 2012 Regulatory Impact Assessment for the revised annual PM NAAQS (U.S. EPA, 2012). These results confirm that, even in places where background air is relatively clean, small changes in air pollution concentration can have population health impacts.

As these studies show, there is no convincing evidence to date showing that there is any threshold below which such adverse effects of PM air pollution will not occur. This lack of a threshold of effects indicates that any reduction in air pollution can be expected to result in commensurate health benefits to the public at ambient levels.

With respect to $PM_{2.5}$ from power plants, my recent studies, and those by others, have also found that long-term exposure to combustion-related fine particulate air pollution is a particularly important environmental risk factor for cardiopulmonary and lung cancer mortality. Air pollutants associated with fossil fuel combustion (e.g., from oil, coal, and natural-gas-fired power plants) have well-documented adverse human health effects. The health impact is particularly high for particulate matter from fossil-fuel-burning facilities, such as coal burning, which has been associated with an ischemic heart disease mortality risk that is roughly five times that of the average for $PM_{2.5}$ particles in general (Thurston *et al.*, 2016b), and more damaging per $\mu g/m^3$ than $PM_{2.5}$ from other common sources (Figure 6).

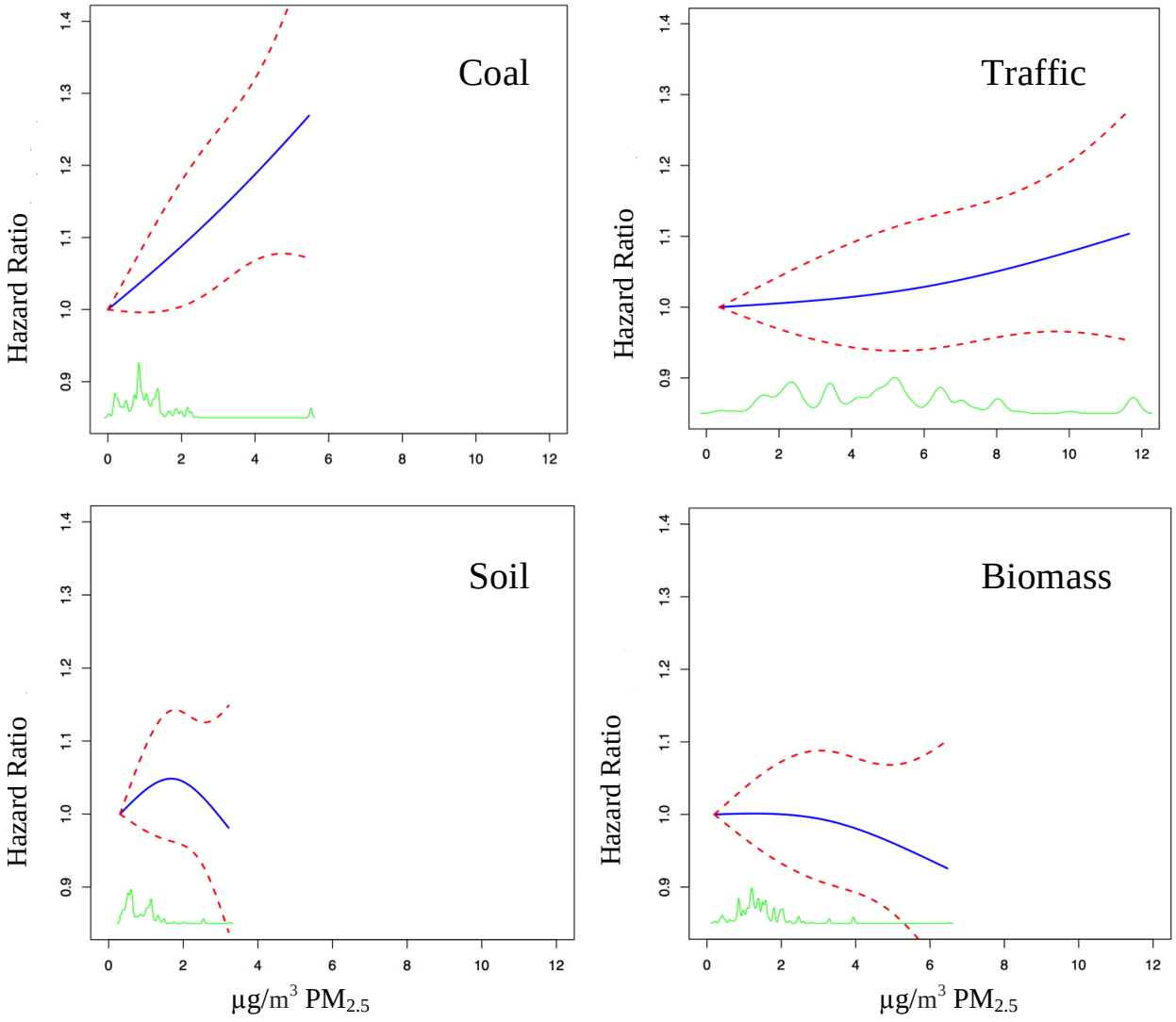

Figure 6. Concentration-response curve (solid lines) and 95% confidence intervals (dashed lines) for source-specific $PM_{2.5}$ mass in the US American Cancer Society (ACS) Cohort. (Thurston et al., 2016b).

Thus, this new study, combined with past studies of US mortality and source-specific $PM_{2.5}$ (e.g., Ozkaynak and Thurston, 1987) indicate that the estimates provided here are conservative underestimates of the health benefits that would result from these proposed emissions controls, because the particles resulting from coal-combustion that will be eliminated are apparently far more toxic to human health than the average $PM_{2.5}$ mass, when considered on per $\mu g/m^3$ mass basis. Thus, by assuming in this report that the toxicity of the particles controlled are the of same toxicity as other particles (including, for example, wind blown soil), the estimates provided for the numbers and monetary valuations of the human health benefits of the BART controls are very conservative.

Sulfur oxide (SOx) exposures have also been associated with adverse health effects, in addition to leading to the secondary formation of $PM_{2.5}$ in the atmosphere. As concluded in the most recent U.S. EPA Risk and Exposure Assessment Report for SO_2 (EPA-452/R-09-007), research studies have provided scientific evidence that is sufficient to infer a similar relationship to also exist between short-term (e.g., daily) SO_2 exposure and adverse effects on the respiratory system. This finding of a causal relationship between SO_2 exposure and increased respiratory morbidity is supported by a large body of recent epidemiologic evidence, as well as by findings from human and animal experimental studies. These epidemiologic and experimental studies encompass a number of endpoints, including ED visits and hospitalizations, respiratory symptoms, airway hyperresponsiveness, and lung function (U.S. EPA, 2009).

Overall, there is a consistency between the epidemiologic study associations and experimental study results, supporting the conclusion that 1) there is indeed a cause-effect relationship between air pollution and negative health effects; and, 2) there is no known threshold below which no effects are experienced. Thus, reductions in air pollution result in commensurate improvements in public health, as provided in this report.

METHODS

The U.S. EPA-approved Environmental Benefits Mapping and Analysis Program (BenMAP) is a Windows-based computer program that uses a Geographic Information System (GIS)-based method to estimate the health impacts and economic benefits occurring when populations experience changes in air quality (Abt Associates, 2010; U.S. EPA, 2015). Analysts have relied upon BenMAP to estimate the health impacts from air quality changes at the city and regional scale, both within and beyond the U.S. A copy of my BenMAP certification is attached as Exhibit T-2. Some of the purposes for which BenMAP has been used include the following:

- Generation of population/community level ambient pollution exposure maps;
- Comparison of benefits across multiple regulatory programs;
- Estimation of health impacts associated with exposure to existing air pollution concentrations;
- Estimation of health benefits of alternative ambient air quality standards.

BenMAP is primarily intended as a tool for estimating the health impacts, and associated economic values, associated with changes in ambient air pollution, as we apply it here. It accomplishes this by computing health impact functions that relate a change in the concentration of a pollutant with a change in the incidence of a health endpoint.

Inputs to health impact functions in this work included:

- The change in ambient air pollution level (as provided by Dr. Andrew Gray, of Gray Sky Solutions);
- Pollutant health effect estimates (based upon the scientific literature and present EPA practice);
- The exposed population, on a county basis, as provided in the BenMAP model; and,
- The baseline incidence rate of the health endpoint, on a county basis, as provided in the BenMAP model.

For example, in the case of a premature mortality health impact function, the BenMAP calculation can be represented, in a simplified form, as:

Mortality Change = (Air Pollution Change) * (Air Pollution Mortality Effect Estimate) * (Mortality Incidence)* (Exposed Population)

- **Air Pollution Change**. The air quality change is calculated as the difference between the starting air pollution level, also called the baseline, and the air pollution level after

some change, such as that caused by a regulation. In the case of particulate matter, this is typically estimated in micrograms per meter cubed ($\mu g/m^3$). In this analysis, these concentrations were provided on a county-by-county population weighted centroid basis.

• **Mortality Effect Estimate**. The mortality effect estimate is an estimate of the percentage change in mortality due to a one unit change in ambient air pollution. Epidemiological studies provide a good source for effect estimates.[4] In this Report, since the choice of mortality effect study has such a large influence on the valuation of the adverse health impacts avoided by applying EPA's proposed emission limits, I have presented (in Table 1) several BenMAP estimates for the mortality effect estimate, ranging from the lower end of estimates (Krewski et al., 2009), the higher end (Laden et al, 2007), and an intermediate estimate (Lepeule et. al, 2012). However, for breakdowns in adverse effects, in order to show the distribution of the effects benefits of EPA's proposed BART controls (e.g., between states, power plants, or metropolitan areas, as in Tables 2-4), I present results using only the low mortality effect estimate (Krewski et al, 2009) to simplify comparisons. This conservative (lowest benefit estimate) choice of the ACS Cohort studies to evaluate mortality benefits of EPA's proposed emissions reductions is consistent with the estimate used by EPA in the agency's prior nationwide analysis of the health benefits of Best Available Retrofit Technology determinations under the Regional Haze Regulations.[5] This choice of a specific mortality study does not affect the *relative* comparisons between states, power plants, etc., which would remain the same irrespective of mortality effect estimate choice. It should be noted that, if I instead used the higher mortality per $\mu g/m^3$ $PM_{2.5}$ effect estimates from the other two studies in Table 1 (which are also scientifically supportable), the dollar valuation of health benefit estimates in Tables 2 thru 4 would be approximately 2.2 times higher using the Lepeul et al. study mortality effect estimate, or approximately 2.8 times higher using the Laden et al. study mortality effect estimate, across the board. However, the ratios of the *relative* impacts across categories would be unaffected by the choice of mortality

[4] When multiple epidemiological studies are available in BenMAP for a health outcome, multi-study pooled estimates have been made, following recent EPA practice (e.g., USEPA, 2012), and as delineated in Table 1.

[5] EPA, Regulatory Impact Analyis for Final Clean Air Visibility Rule of the Guidelines for Best Available Retrofit Technology (BART) Determinations Under the Regional Haze Regulations, EPA-452/R-05-004 (June 2005), *available at* http://www.epa.gov/oar/visibility/pdfs/bart_ria_2005_6_15.pdf.

impact study effect estimate.

- **Mortality Incidence**. The mortality incidence rate is an estimate of the average number of people that die in a given population over a given period of time, as provided in BenMAP. For example, the mortality incidence rate might be the probability that a person will die in a given year.
- **Exposed Population**. The exposed population is the number of people affected by the air pollution reductions required under EPA's BART proposal, based on Census data for each county within BenMAP.

For this work, population-weighted centroid $PM_{2.5}$ concentration impacts from each source in each county in the fourteen study states (Alabama, Arkansas, Colorado, Illinois, Indiana, Kansas, Kentucky, Louisiana, Mississippi, Missouri, New Mexico, Oklahoma, Tennessee, and Texas) were determined by Andrew Gray for the (1) existing emissions; and (2) controlled emissions scenarios based on EPA's BART Proposal, respectively. *See* Gray, Visibility and Health Modeling, Technical Support Document to Comments of Conservation Organizations (May 5, 2017), EPA Docket No. EPA-R06-OAR-2016-0611. The arithmetic difference between the two scenario results were calculated (on a plant-by-plant and county-by-county basis) as the concentration reduction associated with the BART controls, for each plant and county in the study area modeled by Dr. Gray. As outlined in more detail by Dr. Gray in his report, CALPUFF air dispersion modeling was used to estimate long-term (three-year modeled average) fine PM concentrations at the 837 county receptors within the CALPUFF modeling domain for both the 2001-2004 emissions baseline and for the proposed BART control emission scenario. Postprocessing of the CALPUFF results was performed to sum the modeled sulfate, nitrate, and PM2.5 at each receptor in order to estimate the total fine PM concentration at each receptor, as contributed by each source, under both baseline and the control scenarios.] These values were entered into BenMAP to estimate the health benefits, and their dollar valuations, associated with EPA's BART controls on a county-by-county basis for each of nine electrical generating power plant sources. The results for the nine power plants proposed for BART control by the EPA were then summed on a cumulative basis (Table 1, with both numbers and valuations, by cause). Furthermore, to allow an indication of the plant-by-plant and spatial distribution of the health and economic benefits from EPA's BART proposal, the health benefit valuations (summed over all causes, as dollars) were also calculated on a state-by-state (Table 2),

plant-by-plant (Table 3), and metropolitan area-specific (Table 4) basis, providing insight into the relative health impacts by specific sources to specific areas benefitting from the pollution control FIP. The Appendix to this report provides a complete breakdown of the annual health benefits associated with the application of EPA's proposed emission limits by individual power plant and health effect.

RESULTS

Using the above-described EPA BenMAP methodology-based analysis, I conservatively estimate the total public health-based economic benefits associated with reductions in ambient $PM_{2.5}$ concentrations as a result of applying EPA's BART control determinations to the 18 individual Texas EGUs (as displayed in Table 1 for all nine electric generating stations, and all states considered, combined) to be between roughly $6.7 billion and nearly $17 billion per year, overall, primarily depending on the epidemiological study used to determine the $PM_{2.5}$ mortality impacts (*i.e.*, Krewski et al. (low), Lapieule et al. (mid), or Laden et al. (high)). These impacts reflect the range of potential mortality effects associated with the proposed EPA FIP, depending on the particular study used to estimate the effect per $\mu g/m^3$ $PM_{2.5}$ exposure. Further breakdowns of Table 1's estimates using only the Krewski et al. study (*i.e.*, the low mortality effect estimate) to estimate total mortality impacts are provided in Tables 2 through 4 of this report: *i*) by electric generating power plant (i.e., for each of the nine power plants over all areas modeled by Dr. Gray); *ii*) for all power plant generating unit impacts collectively by State of impact (i.e., in Alabama, Arkansas, Colorado, Illinois, Indiana, Kansas, Kentucky, Louisiana, Mississippi, Missouri, New Mexico, Oklahoma, Tennessee, and Texas); and, *iii*) for all power plant generating unit impacts collectively by major metropolitan impact area.

As seen in Table 1, the numbers of adverse health events avoided by application of EPA's proposed SO_2 emission limits are dominated by the morbidity events, such as respiratory symptoms, restricted activity days, and work loss days. In contrast, the dollar valuation of the adverse health events are largely dominated, as would be expected, by the more severe health outcomes, including myocardial infarctions (heart attacks), chronic bronchitis, and (especially) deaths. As shown in Table 2, on a state-by-state basis, the largest health benefits go to the state in which the power plants are operating (Texas), but, because this pollution can be carried so far downwind, nearly one half of the health benefits would accrue in other (downwind) states. On a power plant basis (Table 3), it is seen that large health benefits are derived from controlling each

the nine plants EPA proposes to regulate under this FIP, with the greatest benefits being derived from controlling the Big Brown, Martin Lake, and Monticello plants. Table 4 makes clear that urban areas in Texas would receive the largest health benefits from the proposed BART emission controls at these generating stations, but that the benefits stretch long distances downwind, with still very large health benefits in cities as far away as Illinois.

Table 1. Annual Multi-State Human Health Effects and Monetary Valuations Associated With the $PM_{2.5}$ Air Pollution Avoided by Applying EPA's Proposed BART Controls for Texas Sources

Health Endpoint	**Expected Number Per Year Avoided***	**Total Dollar Valuation (2010$)****
Respiratory Hospital Admissions (Kloog et al., 2012; Zanobetti et al., 2009)	125[a]	$3,966,000
Cardiovascular Hospital Admissions (Bell et al., 2008; Peng et al., 2008; Peng et al., 2009; Zanobetti et al., 2009)	125[a]	$4,733,000
Acute Bronchitis (Dockery et al., 1996)	1317	$633,000
Acute Myocardial Infarction, Nonfatal (Pope et al., 2006; Sullivan et al., 2005; Zanobetti et al., 2009; Zanobetti & Schwartz, 2006)	80[b]	$10,094,000[a]
Emergency Room Visits (Glad et al., 2012; Mar et al., 2010; Slaughter et al., 2005)	381[b]	$162,000[a]
Asthma Exacerbation Symptoms (Mar et al., 2004; Ostro et al., 2001)	24,818[b]	$1,434,000
Upper Respiratory Symptoms (Pope et al., 1991)	23,915	$795,000
Lower Respiratory Symptoms (Schwartz and Neas, 2000)	16,767	$352,000
Minor Restricted Activity Days (Ostro & Rothschild, 1989)	625,525	$42,754,000
Work Days Lost (Ostro et al., 1987)	105,853	$15,803,000
Chronic Bronchitis (Abbey et al., 1995)	521	$147,152,000[c]
Mortality, All Causes (Krewski et. al, 2009)	678	$6,518,235,000
Mortality, All Causes (Lepeule et. al, 2012)	1541	$14,823,929,000
Mortality, All Causes (Laden et al., 2007)	1760	$16,921,843,000

* Rounded to nearest whole number.

** Rounded to nearest $1000.

a Pooled effects with averaging approach, as per EPA BenMAP default setting.

b Pooled effects with random/fixed effects approach, as per EPA BenMAP default setting.

c Pooled effects with summation approach, as per EPA BenMAP default setting.

Table 2. State-By State Total Valuation of Annual Health Benefits of EPA Proposed BART Controls Applied to the Nine Power Plants At Issue* (Applying Krewski et al., 2009 for mortality)

State	Total Dollar Valuation (2010$)**
AL	$57,080,000
AR	$522,356,000
CO	$5,564,000
IL	$46,516,000
IN	$12,432,000
KS	$152,556,000
KY	$35,415,000
LA	$492,830,000
MS	$241,108,000
MO	$324,832,000
NM	$38,796,000
OK	$771,304,000
TN	$149,283,000
TX	$3,896,042,000
Total	$6,746,113,000

* Big Brown, Coleto Creek, Fayette, Harrington, JT Deely, Martin Lake, Monticello, Parish, and Welsh.

** Rounded to nearest $1000.

Table 3. Plant-By Plant Total Valuation of Annual Health Benefits of EPA Proposed BART Controls (Applying Krewski et al., 2009 for mortality)

Electric Generating Station	Total Dollar Valuation (2010$)*
Big Brown	$1,617,952,000
Coleto Creek	$261,901,000
Fayette	$495,331,000
Harrington	$153,627,000
JT Deely	$508,409,021
Martin Lake	$1,135,234,000
Monticello	$1,553,080,000
Parish	$816,736,000
Welsh	$203,842,000
Total	$6,746,113,000

* Rounded to nearest $1000.

Table 4. Total Valuation of Annual Health Benefits of EPA Proposed BART Controls for Selected Metropolitan Areas (Applying Krewski et al., 2009 for mortality)

City (Counties)	**Total Dollar Valuation All 9 Plants (2010$)***
Austin, TX (Hayes, Travis, Williamson)	$182,849,000
Dallas, TX (Colin, Dallas, Ellis, Rockwall)	$623,296,000
Ft. Worth, TX (Johnson, Tarrant)	$369,004,000
Houston, TX (Brazoria, Chambers, Fort Bend, Galveston, Harris)	$606,467,000
San Antonio, TX (Bexar, Comal, Guadalupe)	$325,461,000
Little Rock, AR (Lonoke, Pulaski, Saline)	$90,863,000
Kansas City, KS (Johnson, Wyandotte, Cass, Clay, Jackson, Platte)	$6,670,000
New Orleans, LA (Jefferson, Orleans, Plaguemines, St. Bernard, St. Charles, St. John the Baptist)	$56,435,000
Jackson, MS (Hinds, Madison, Rankin)	$39,942000
Oklahoma City, OK (Canadian, Cleveland, Logan, Oklahoma)	$185,587,000
Tulsa, OK (Creek, Osage, Tulsa, Wagoner)	$156,516,000
Nashville, TN (Davidson, Fsumner, Williamson, Wilson	$2,911,000

* Rounded to nearest $1000.

In addition to reflecting a conservative (i.e., low) mortality effects estimate, these overall health impact counts and their dollar valuations are conservative estimates of the health benefits after the application of the proposed BART controls at the affected power plant units for a number of reasons, including: (a) additional health impacts not modeled in this analysis attributable to co-reductions in other pollutants (*e.g.*, gaseous SO_2) are not included here; (b) consideration of health impacts only for the ages of the exposed populations that were considered in the epidemiological studies on which these analyses were based; (c) there are either no health impact studies or no dollar valuation available for many health outcomes thought to be adversely affected by air pollution, such as effects of air pollution on birth outcomes; and (d) in Tables 2-4 we have applied the low estimate of the mortality benefits (whereas applying the other two studies noted would roughly double or triple the estimates in Tables 2-4, respectively) . Thus, while all air pollution control costs associated with application of EPA's proposed BART controls can be estimated, these estimates of the health benefits and their monetary valuations are only available for a subset of likely health impacts from air pollution. This, in addition to prior discussion of the likely higher toxicity of particles from coal-fired power plants, means that my analysis is very conservative, and likely underestimates the health and monetary benefits of applying EPA's BART emission limits to the affected Texas power plant units.

CONCLUSIONS

Even applying conservative estimates and assumptions, the health benefits and valuations derived from the application of EPA's BART control determination to the 9 Texas electric generating power plants at issue are substantial. Moreover, these benefits and their valuations accrue each and every year those controls are operational. Accordingly, ten years from the compliance date, the health benefits and valuations of proposed controls will be roughly ten times the values provided in Tables 1 through 4, before adjustment for a discount rate and future affected population growth, as appropriate. Similarly, these benefits and their valuations are lost (not accrued) each and every year that application of the EPA's BART controls are delayed. Thus, even a delay of just a few months carries the risk of substantial, and irreparable, harm to public health. As demonstrated above, those public health impacts have an associated and quantifiable adverse economic impact. Thus, it is reasonable to conclude that any delay implementing EPA's Regional Haze BART controls for Texas will only exacerbate the substantial, and irreparable, harms to public health that have already been incurred to date by the operation of these electric generating units.

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