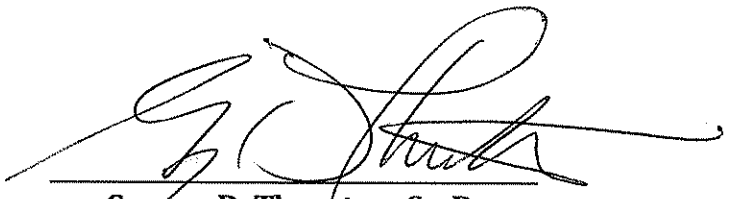
RE: ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 49 [EPA-R09-OAR-2013-0009; FRL-]

Approval of Air Quality Implementation Plans; Navajo Nation; Regional Haze Requirements for Navajo Generating Station; Supplemental Proposal

WRITTEN REPORT OF GEORGE D. THURSTON
REGARDING THE PROPOSED NAVAJO GENERATING PLANT EPA RULEMAKING

December 12, 2013



George D. Thurston, Sc. D.
3 Catherine Court
Chester, NY 10918

I, George D. Thurston, declare and state as follows:

INTRODUCTION AND BACKGROUND

1. My name is George D. Thurston, and I am Professor of Environmental Medicine at the New York University (NYU) School of Medicine. My environmental health business address is 3 Catherine Ct., Chester, NY 10918. I am over the age of 18 years and I am competent to testify concerning the matters in this affidavit.

2. 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 25 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 years 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 1.

3. This declaration is submitted in response to the U.S. EPA's request for comments on its proposed options for BART at Navajo Generating Station coal-fired power plant ("Navajo Power Plant") including the Technical Work Group (TWG) Alternative to BART technology to the Navajo Power Plant.

4. 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 by excess emissions of the nitrogen oxides, ozone, and fine particulate matter that will result if the application of BART technology is delayed at the Navajo Power Plant. For purposes of this report, "BART technology" and associated references to pollutant reductions

with BART pollution controls means 89% reduction in nitrogen oxides from all three units of Navajo Power Plant as proposed by EPA in its initial BART proposal.

5. This report documents how emissions from the Navajo Power Plant's units contribute to the serious and well-documented adverse human health effects known to be associated with exposure to air pollution, including that from the fossil-fuel fired utility plants considered here. 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 other expert reports submitted by Andrew 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 human health impacts that would occur each year effective BART pollution controls are delayed, as well as the annual economic valuation of those unavoided health impacts.

HEALTH IMPACTS FROM POWER PLANTS LIKE THE NAVAJO POWER PLANT

6. The adverse health consequences of breathing air pollution from sources such as 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 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 daily 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.

A. Fine Particulates

7. Fine Particulate Matter (PM) is among the key air pollutants caused by 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 the past decade. 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. For example, after it is released from a smokestack, gaseous NOx is chemically converted in the atmosphere to become nitrate PM. These particles are much smaller than most natural particles found in the atmosphere, such as windblown dust, commonly having an aerodynamic diameter of less that 1.0 micrometer (μm) – a fraction of the diameter of a human hair. As such, because of their small size, these combustion particles can avoid the body's normal defenses that have developed against larger particles, such as impaction and clearance in the nose and throat, and these combustion particles can therefore have a far greater adverse effect on health than most other particles in the air.

8. The conclusion that power plant particle pollution is one of the more toxic types of particles that we breathe is supported by the facts that combustion particles also have physiochemical characteristics different from other more "natural" particles, such as wind-blown soil. In particular, these power plant particles are enriched in toxic metals, such as arsenic and cadmium, as well as in transition metals, such as iron and vanadium, that can cause damaging oxidative stress in lung cells (e.g., Costa et al, 1997; Dreher et al, 1997, and; Lay et al, 1999). This may also be especially true in the case of power plant particles because of the co-presence of acidic sulfates, such as sulfuric acid, that can make these transition metals even more bio-available and potent to damage the lung (e.g., Chen et al. 1990, Gavett et al., 1997). Moreover, since these power plant PM penetrate deep into the lung where they are not easily cleared, and can therefore reside for long times, they have greater "opportunity" to cause significant damage to the lung and to the human body. Thus, power plant air pollution is cause for special concern, and adds urgency to the need for reductions in the amounts of this pollution emitted into our air.

9. In addition to lung damage described above, 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, 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 ug/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 ug/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 show qualitatively consistent acute cardiac effects, such as the Sullivan et al. (2005) study of acute myocardial infarctions in King County, Washington; Pope et al (2006) conducted a case-crossover study to analyze ischemic events in 12,865 patients who lived on the Wasatch Front in Utah, finding that $PM_{2.5}$ elevated by 10 microg/m3 was associated with increased risk of acute ischemic coronary events (unstable angina and myocardial infarction) equal to 4.5% (95% confidence interval, 1.1 to 8.0); Zanobetti and Schwartz (2006) study of hospital admissions through emergency department for myocardial infarction (ICD-9 code 410): and, Zanobetti et al. (2009) 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. Of these, the Peters study is especially highly relied upon because individual cases were more definitively confirmed by detailed chart reviews and patient interviews that were conducted by trained research personnel (Peters et al., 2001).

10. 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.* (2000) 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.

11. 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 month, and year after year, until they account for thousands of needless daily deaths from air pollution in the U.S. each year.

12. Epidemiologic research conducted on U.S. residents has indicated that acute short-term exposures to PM air pollution, are associated with increased risk of mortality. For example, a nationwide time-series statistical analysis of daily death counts by the Health Effects Institute ("HEI," 2003) examined mortality and PM_{10} air pollution (a subset of particulate matter air pollution that is less than 10 μm in diameter, including $PM_{2.5}$) in 90 cities across the United States, finding that, for each increase of 10 $\mu g/m^3$ in daily PM_{10} air pollution concentration, there is an associated increase of approximately 0.3% in the *daily* risk of death by the public. Indeed, and I concur, the most recent U.S. EPA Particulate Matter Integrated Science Assessment unequivocably 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.*"[1]

13. In addition to the acute health effects associated with daily PM pollution, 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 (that was a 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 (H). Air quality was averaged over the period of study in order to study long-term (chronic) effects. As shown in Figure 7, 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.

[1] U.S. Environmental Protection Agency (2009): Integrated Science Assessment for Particulate Matter (Final Report), Washington, DC, EPA/600/R-08/139F, at 2-10, 2-11 (emphasis in original), available at http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=216546

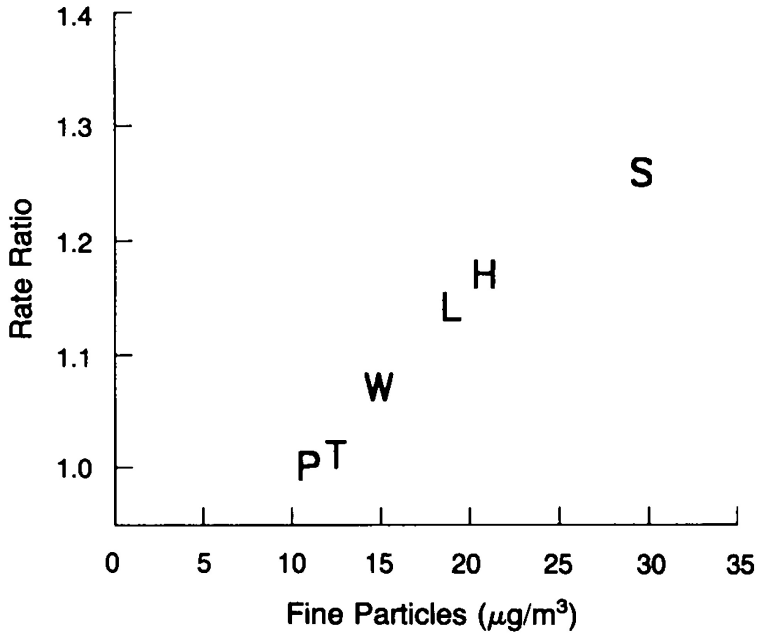


Figure 1. The Harvard Six-Cities Study showed that the lifetime risk of death increased across 6 U.S. cities as the average fine PM levels increased. (Source: Dockery *et al.*, 1993).

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, 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).

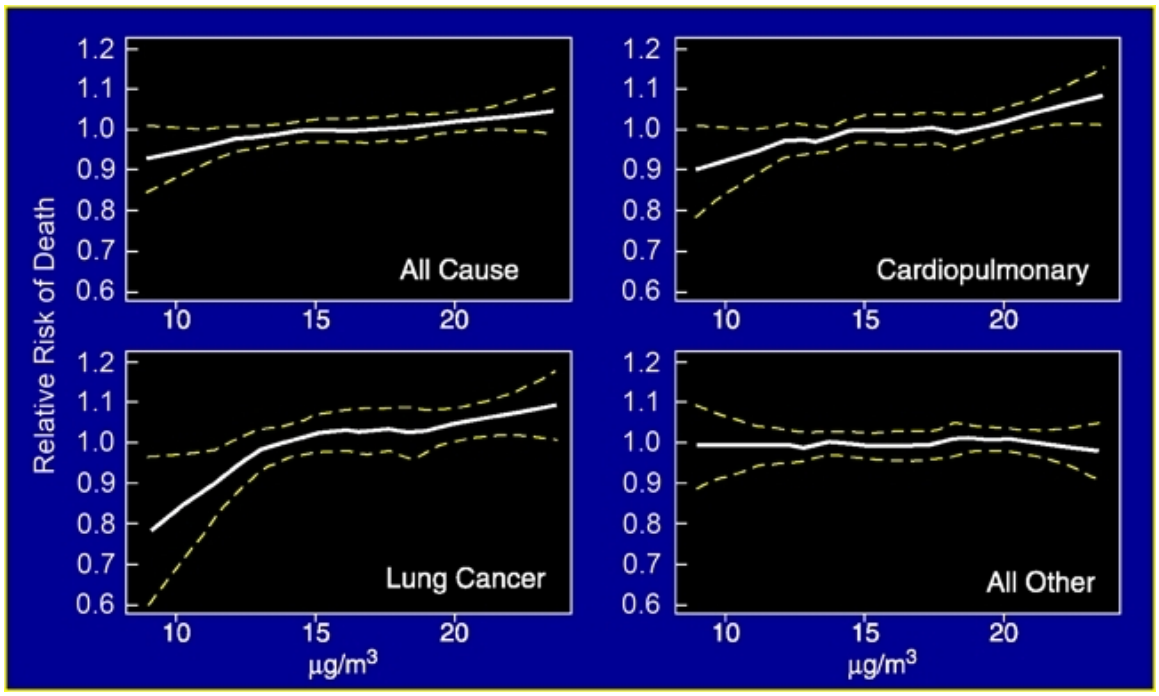


Figure 2. Cardiopulmonary and Lung Cancer Mortality Risks Increase Monotonically with Exposure to Long-Term Fine PM
(Adapted from: Pope, Burnett, Thun, Calle, Krewski, Ito, and Thurston, 2002)

14. 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.

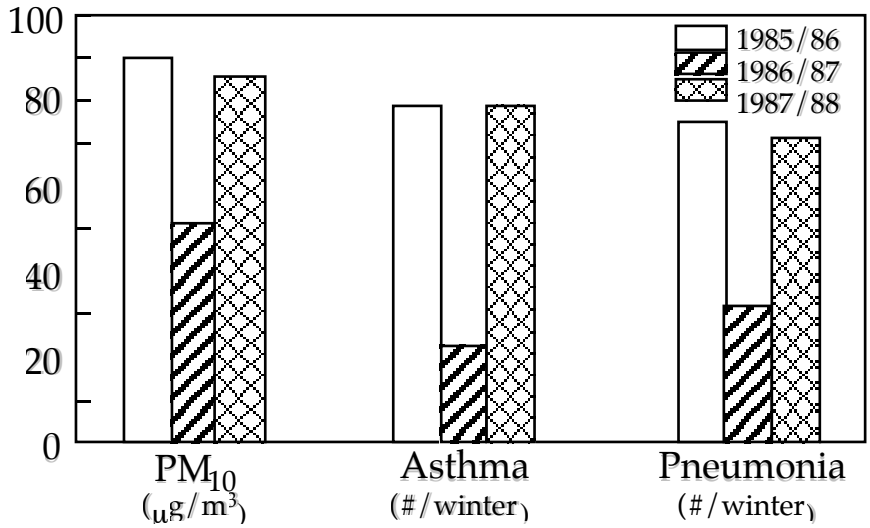


Figure 3. Decreasing PM pollution lowered the number of children's hospital admissions (Source: Pope, 1989).

15. 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 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 was 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."[2] "Consistently," Dr. Laden said, "in the cities where there was the most cleanup, there was also the greatest decrease in risk of death."

16. 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 $\mu g/m^3$.

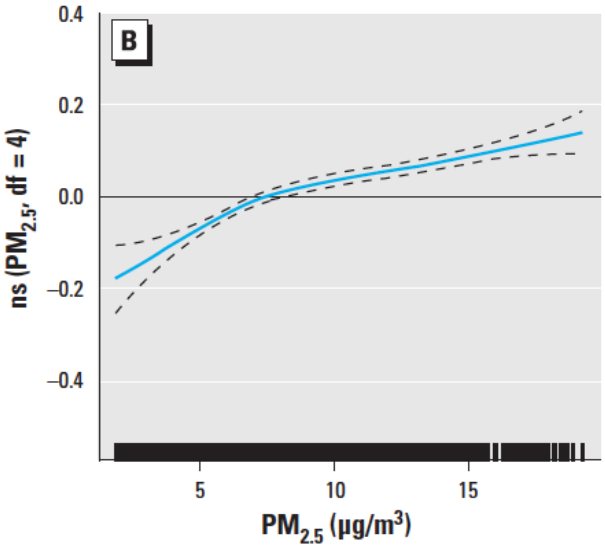


Figure 4. Cardiovascular Mortality Risk vs. $PM_{2.5}$ exposure (solid line) and 95% CIs (dashed lines), showing increasing risk of death with increasing $PM_{2.5}$, even at very low ambient levels of $PM_{2.5}$ air pollution (from Crouse et al., 2012).

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

17. Although published too late to have been considered by the U.S. EPA in their initial BART analysis in early 2013, the 2012 Crouse 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 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.**

18. 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.

B. Ozone

19. O_3 is another important air pollutant resulting from nitrogen oxide and hydrocarbon power plant emissions that adversely affects human health. Ozone is a highly irritating gas that is formed in the atmosphere in the presence of sunlight from other "precursor" air pollutants, including NO_x and hydrocarbons that are emitted by combustion sources such as fossil fuel burning power plants. The adverse health consequences of breathing ozone are serious and well documented. This documentation includes impacts demonstrated in controlled chamber exposures of humans and animals, and observational epidemiology showing consistent associations between ozone and adverse impacts across a wide range of human health outcomes.

20. The noxious nature of ozone is also evidenced by the way it visibly "eats away" at materials such as rubber, an elastic substance sharing characteristics with human lungs. Indeed, in the early years of air pollution monitoring, the number of cracks in a stretched rubber band left outdoors for weeks was used as an index of the ozone concentration in the air. Similarly, ozone has been known to cause fading of certain colors in fabrics because they oxidize the dye, causing "O-fading." As a result, automobile manufacturers today utilize ozone-resistant rubbers, while carpet and drape manufacturers use ozone-resistant dyes (NRC, 1976). In addition, Cass *et al.* (1991) have discussed the importance of protecting works of art from damage due to O_3. Given this evidence of ozone's devastating effects on solid materials, it comes as no surprise that ozone

can also have serious adverse health effects on the more vulnerable human lung.

21. Ozone can irritate the human respiratory system, causing exposed people to cough, feel an irritation in the throat, and/or experience an uncomfortable sensation in the chest area. Ozone has also been shown to reduce the lung's ability to inhale and exhale, thereby making it more difficult for people to breathe as deeply and vigorously as they normally would (*e.g.*, *see* Bates, 1995). Research shows that ozone can also acutely aggravate asthma, and new evidence suggests that it may cause more children to get asthma. When ozone levels are high, people with asthma have more attacks that require a doctor's attention or the use of additional medication. One reason this happens is that ozone makes people more sensitive to allergens, which are the most common triggers for asthma attacks. Ozone can inflame and damage cells that line the human lung, and O_3 has been compared by some to "getting a sunburn on your lungs." Ozone may also aggravate chronic lung diseases, such as emphysema and bronchitis, and can reduce the immune system's ability to fight off bacterial infections in the respiratory system.

22. Among the important adverse effects associated with ozone exposure to asthmatics is the triggering of asthma attacks. The effects of ozone air pollution on children with asthma have been demonstrated in my own research following a group of children at an asthma summer camp located in Connecticut. This study of a group of about 55 moderate to severely asthmatic children showed that these children experienced statistically significant reductions in lung function, increases in asthma symptoms, and increases in the use of unscheduled asthma medications as ozone pollution levels rose. As shown in Figure 5, the risk of a child having an asthma attack was found to be approximately 40 percent higher on the highest ozone days than on an average study day (Thurston *et al.*, 1997). Consistent with other research in this area, there is no indication in this plot of a threshold concentration below which children with asthma are safe from the effects of ozone increases.

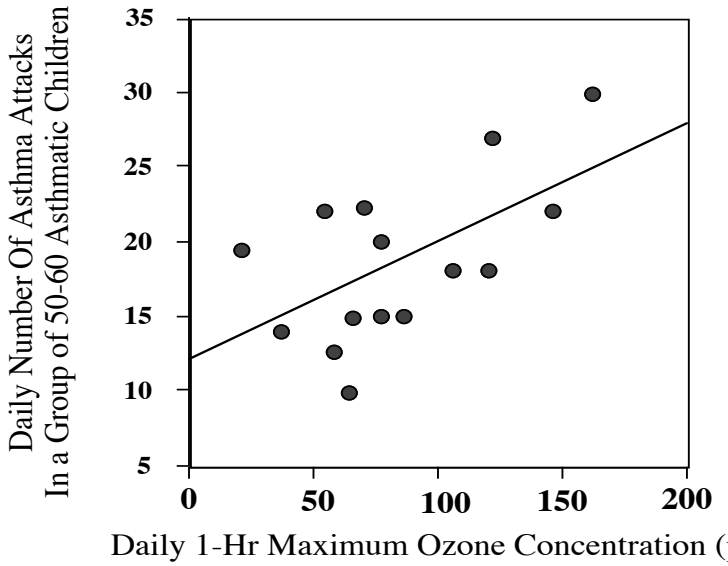


Figure 5. The number of asthma attacks among children at an "Asthma Camp" in Connecticut increase as the ozone levels rise (Source: Thurston *et al.*, 1997)

23. These asthma camp results have been confirmed by a larger study published in the Journal of the American Medical Association (JAMA). Gent *et al.* (2003) presented a cohort study of asthmatic children from the New Haven, CT area, including 130 children who used maintenance medications for asthma and 141 children who did not. The more severe asthmatics were identified as those using maintenance medication. For these severe asthmatics, the study found that the level of O_3 exposure was significantly associated with worsening of symptoms and an increase in the use of rescue medication. Each 50 parts per billion (ppb) increase in 1-hour average O_3 was associated with an increased likelihood of wheezing (by 35%) and chest tightness (by 47%). The findings indicate that asthmatic children are particularly vulnerable to ozone, even at pollution levels below the U.S. EPA air quality standards.

24. My own research has also shown ozone air pollution to be associated with diminished lung function in non-asthmatic healthy children at a YMCA summer camp in a pristine area in the Kittatinny Ridge, in the northwestern part of the state (Spektor *et al.*, 1988a). Similarly, in the summer of 1988, Berry *et al.* (1991) conducted a field health study at two summer day camps in suburban-central New Jersey. Thirty-four campers and counselors had daily lung function tests, and it was found that the campers had a statistically significant decrease in peak expiratory flow rate associated with increasing

ozone concentrations, indicating an acute loss in the children's ability to inhale and exhale after ozone exposure.

25. The adverse effect of exposure to ozone in ambient air on the lungs of individuals has been demonstrated in studies that I have conducted in the State of New York, as well. For example, respiratory function damage was demonstrated in a study I co-authored of 30 healthy adult non-smokers engaged in a regular daily program of outdoor exercise in Tuxedo, NY during the summer of 1985 (Spektor *et al.*, 1988b). All measured health indices showed statistically significant O_3-associated decreases in the lung function of the runners as ozone levels increased. More recently, using lung bronchoscopy (which allows a visualization of the main tubes of the lungs, by means of a flexible lighted instrument introduced through the vocal cords and windpipe) and broncho-alveolar lavage (BAL, or a washing of the lining of the lung), Kinney *et al.* (1996) examined some 19 normal volunteer joggers from Governors Island, NY. The joggers exercised in the afternoon during the 1992 summer season. These results indicate a significant inflammatory response in the lungs of recreational joggers in New York City exposed to regional ozone and associated co-pollutants during the summer months.

26. Airway inflammation in the lung is among the serious effects that have also been demonstrated by controlled human studies of ozone at levels typically experienced by most Americans. Airway inflammation is especially problematic for children and adults with asthma, as it makes them more susceptible to having asthma attacks, consistent with the asthma camp results discussed above. For example, controlled human studies have shown that prior exposure to ozone enhances the reactivity of asthmatics to aeroallergens, such as pollens, which can trigger asthma attacks (*e.g.*, *see* Molfino *et al.*, 1991).

27. The increased inflammation of the lung, and diminished immune system effects associated with ozone air pollution can also make the elderly more susceptible to pneumonia, a major cause of illness and death in this age group. Both *in vivo* and *in vitro* experimental studies have demonstrated that O_3 can affect the ability of the immune system to defend against infection. Increased susceptibility to bacterial infection has been reported in mice at below 80ppb ozone for a single 3-hr exposure (Ehrlich *et al.* 1977). Related alterations of the pulmonary defenses caused by short-term exposures to O_3 include impaired ability to inactivate bacteria in rabbits and mice (Coffin and Gardner 1972; Ehrlich *et al.* 1979) and impaired

macrophage defense mechanisms in the lung (Dowell *et al.* 1970; Goldstein *et al.* 1971; McAllen *et al.* 1981; Amoruso *et al.* 1981). Thus, the biological plausibility of the adverse air pollution health effects associations found by epidemiological studies is supported by a body of controlled exposure animal studies.

28. The O_3 - morbidity associations indicated by the above-presented epidemiological studies are also supported by a large body of data from controlled human exposure studies that give consistent and/or supportive results, and that have demonstrated pathways by which ozone can damage the human body when breathed. Clinical studies have demonstrated decreases in lung function, increased frequencies of respiratory symptoms, heightened airway hyper-responsiveness, and cellular and biochemical evidence of lung inflammation in healthy exercising adults. For example, in controlled exposure studies, McDonnell *et al.* (1991) and Devlin *et al.* (1991) found that prolonged controlled exposures of exercising men to levels of ozone common in present-day U.S. (only 80 ppb) resulted in significant decrements in lung function, induction of respiratory symptoms, increases in nonspecific airway reactivity, and cellular and biochemical changes in the lung.

29. Ozone exposure has also been shown to have adverse effects on athletic performance. Epidemiological evidence compiled more than three decades ago suggested that the percentage of high school track team members failing to improve performance increased with increasing oxidant concentrations the hour before a race (Wayne *et al.* 1967). Controlled exposure studies of heavily exercising competitive runners have demonstrated decreased function at 200 to 300 ppb (Savin and Adams 1979; Adams and Schelegle 1983). More recent studies have shown reduced athletic performance at even lower O_3 concentrations. Schlegle and Adams (1986) exposed 10 young male adult endurance athletes to 120, 180, and 240 ppb O_3 while they exercised for 60 minutes. Although all 10 completed the protocol for filtered (clean) air exposure, 1, 5, and 7 of them could not complete it for the 120, 180 and 240 ppb O_3 exposures, respectively, indicating that higher O_3 concentrations made exercising more difficult.

30. Another study considers a broadly relevant case showing the benefits of cleaner air. During the Atlanta Summer Olympics of 1996, traffic-related ozone and PM declined significantly as a result of the alternative mass transportation strategy implemented to reduce road traffic during the Games (Friedman *et al.*, 2001). These improvements were correlated with changes in the rate of children's hospital admissions. Compared to a baseline period, traffic

related ozone and PM_{10} levels declined by 28% and 16%, respectively. Concentrations of both PM and ozone also rose noticeably after the end of the Olympics. The study showed a significant reduction in asthma events associated with these pollution improvements. This study supports the hypothesis that improvements in acute air pollution can provide immediate health benefits. Ozone may also cause permanent lung damage. Repeated short-term ozone damage to children's developing lungs may lead to reduced lung function in adulthood (*e.g.*, *see* Kunzli et al, 1997). In adults, ozone exposure may accelerate the natural decline in lung function that occurs as part of the normal aging process (*e.g.*, *see* Detels, *et al.*, 1987). One important study suggests that long-term ozone exposure can increase the chances that children will develop asthma disease (McConnnell *et al.*, 2002).

31. Ozone has also been shown to have long-term cumulative health effects in the State of New Jersey in a study that included cadets from the U.S. Military Academy at West Point who attended special summer training in Fort Dix, New Jersey. There was a statistically significant drop in forced expiratory volume in 1 sec of 44 ml ($p = .035$), and there were also significant increases in reports of cough, chest tightness, and sore throat at the follow-up clinic visit: a larger decline in long-term mean Forced Expiratory Volume lung function was observed in cadets at Fort Dix, where ozone exposures were the highest (Kinney and Lippmann, 2000).

32. Emergency Room Visits and Hospital Admissions are also increased by O_3 air pollution. Cody *et al.* (1992) analyzed data on New Jersey hospital emergency department (ED) visits for asthma, bronchitis, and finger wounds (a non-respiratory control) for the period May through August for 1988 and 1989, finding that, when temperature was controlled for in a multiple regression analysis, a highly significant relationship between asthma visits and ozone concentration was identified. In addition, a 5-year retrospective study by Weisel *et al.* (1995) of the association between ED visits for asthma with mean ambient ozone levels was conducted for hospitals located in central New Jersey. An association was identified in each of the years (1986-1990), and ED visits occurred 28% more frequently when the mean ozone levels were greater than 60 ppb O_3, as compared to when they were less than 60 ppb O_3.

33. Epidemiological evidence has accumulated over recent years indicating a role of O_3 in daily hospital admissions. As displayed in Figure 11, time-series studies conducted in the U.S. have shown increased risk of hospital admissions (Relative Risk>1.0) at higher O_3 levels, even after accounting for the effects of PM (Schwartz,J., in Health at the Crossroads, 1997). This work has now been expanded to consider 36 cities across the U.S., finding that, during the warm season of the year, the 2-day cumulative effect of a 5-ppb increase in O_3 was an estimated 0.3% increase in the risk of chronic obstructive pulmonary disease admissions, and a 0.4% increase in the risk of pneumonia admissions (Medina-Ramon *et al.*, 2006).

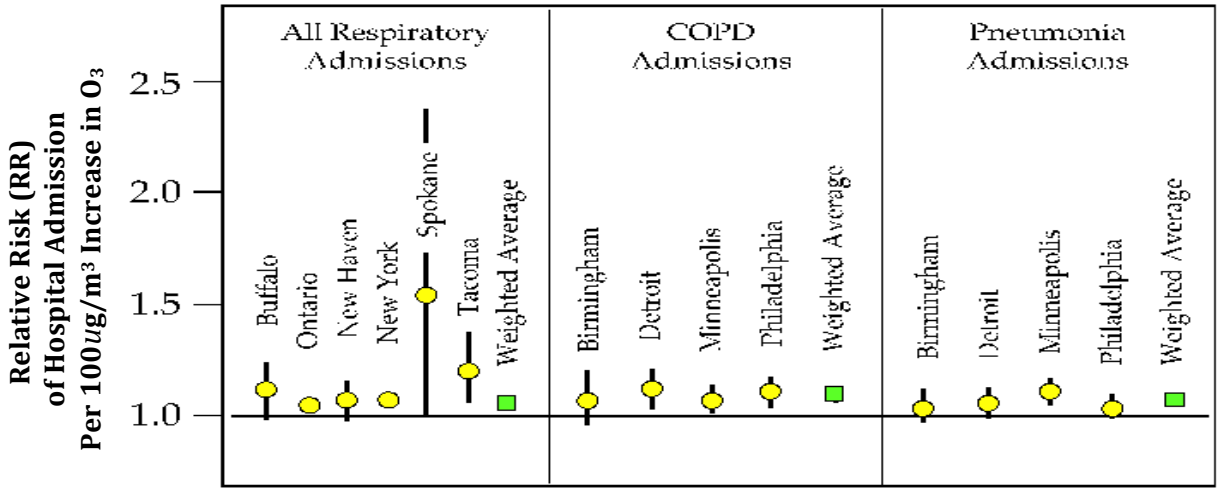


Figure 6. Studies of air pollution in many cities have shown increased risk of respiratory hospital admission (RR >1.0) on days of high ozone air pollution (Source: Schwartz, J. in Health at the Crossroads, 1997).

34. Epidemiological evidence has also accumulated over recent years indicating a role by ozone in daily human mortality. As shown in Figure 7, time-series studies conducted in cities around the world have shown increased mortality (Relative Risk>1.0) at higher ozone concentrations, even after accounting for the mortality effects of PM (Thurston and Ito, 2001).

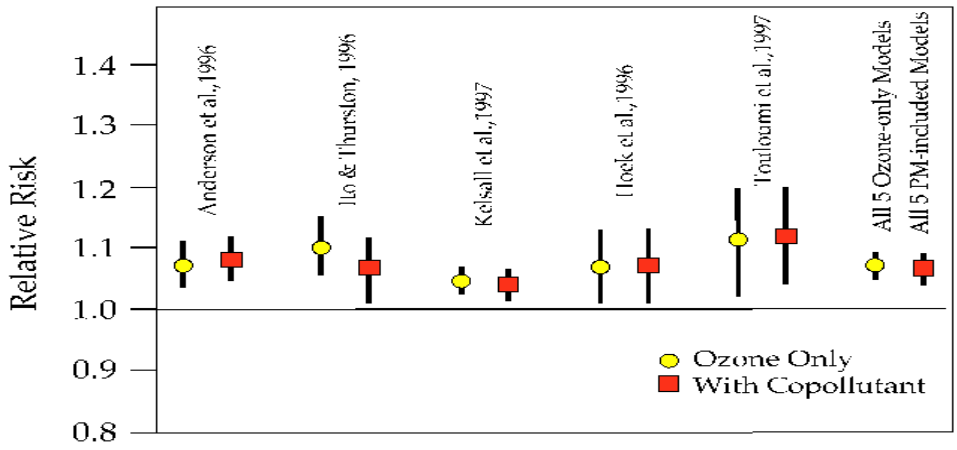


Figure 7. Studies indicate an increased risk of mortality (RR >1.0) at higher ozone concentrations, even after considering the effects of PM. (Source: Thurston and Ito, 2001)

Multi-city analyses have confirmed the ozone-mortality relationship. These include meta-analyses of multiple past ozone studies that show consistent associations between ozone and increases in mortality (Levy et al, 2005; Ito *et al.*, 2005; Bell *et al.*, 2005). In one analysis of some 95 U.S. cities over two decades published in JAMA, Bell et al (2004) showed that, even after controlling for PM and weather, an increase of 10 parts-per-billion in daily ozone pollution was associated with approximately a 0.5% increase in daily risk of death. As discussed earlier, this size percent increase in daily admissions, though small, affects a huge portion of the population and accumulates day after day, week after week, and month after month, so that it accumulates to account for thousands of deaths each year in the U.S.

35. More recently, mortality effects from long-term exposure to ozone air pollution has now been confirmed in a major cohort study. Data from the study cohort of the American Cancer Society Cancer Prevention Study II were correlated with air-pollution data from 96 metropolitan statistical areas in the United States. 448,850 subjects, with 118,777 deaths in an 18-year follow-up period were considered. Data on daily maximum ozone concentrations were obtained from April 1 to September 30 for the years 1977 through 2000. Data on concentrations of fine particulate matter ($PM_{2.5}$) were obtained for the years 1999 and 2000. Associations between ozone concentrations and the risk of death were evaluated with the use of standard and multilevel Cox regression models. In single-pollutant models ozone was associated with the risk of death from respiratory causes. The estimated relative risk of death from respiratory causes that was associated with an increment in ozone concentration of 10 ppb was 1.040 (95% confidence

interval, 1.010 to 1.067). The association of ozone with the risk of death from respiratory causes was insensitive to adjustment for confounders and to the type of statistical model used.

36. Nitrogen oxides exposures have also been associated with adverse health effects, in addition to leading to the formation of $PM_{2.5}$ and ozone. As concluded in a U.S. EPA Risk and Exposure Assessment Report for NOx (EPA-452/R-08-008a), research studies have provided scientific evidence that is sufficient to infer a similar relationship to also exist between short-term (e.g., daily) NO_2 exposure and adverse effects on the respiratory system. This finding is supported by the large body of recent epidemiologic evidence as well as 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, airway inflammation, and lung function (U.S. EPA, 2008).

37. **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 adverse health effects; and, 2) there is no known threshold below which no effects are experienced. Thus, reductions in air pollution are associated with commensurate improvements in public health.**

HEALTH IMPROVEMENTS FROM BART POLLUTION CONTROL TECHNOLOGY AT NAVAJO POWER PLANT

38. 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). 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. 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.

39. 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 running health impact functions, which relate a change in the concentration of a pollutant with a change in the incidence of a health endpoint.

39. Inputs to health impact functions in this work included:

- the reduction in ambient air pollution $PM_{2.5}$ levels (as provided by Andrew Gray, of Gray Skies Solutions), based on the application of BART technology to the Navajo Generating Plant that would reduce stack nitrogen oxides emissions by nearly 90% (from 55,453 TPY to 5,995 TPY NOx).;
- pollutant health effect estimates (based upon the scientific literature and present EPA BenMAP practice);
- the exposed population, on a county and statewide basis, and:
- the baseline incidence rate of the health endpoint, on a county basis.

For example, in the case of a premature mortality health impact function, the BenMAP calculation can be represented, in a simplified representation, 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 ($\mu g/m^3$).
- 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.
- 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 estimated BACT air pollution reductions, based on Census data within BenMAP.

40. Using this 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 initial BART determination to the Navajo power plant units (as displayed in Table 1) to be between $14 million and $35 million per year, overall, primarily depending on the epidemiological study used to determine the $PM_{2.5}$ mortality impacts (i.e., Krewski *et al.* or Laden *et al.*, respectively).

Table 1. Particulate Matter Health Effects and their Monetary Valuations Associated With Each Year that BART Reductions in $PM_{2.5}$ Air Pollution are Not Implemented

Health Endpoint	Expected Number Per Year Avoided*	Total Dollar Valuation (2008$)**
Mortality, All Causes (Krewski et. al, 2009)	2	$13,114,000
Mortality, All Causes (Laden et al., 2007)	5	$33,947,000
Chronic Bronchitis (Abbey et al., 1995)	1	314,000
Acute Myocardial Infarction (Peters et al., 2001)	2	262,000
Work Days Lost (Ostro et al., 1987)	280	$30,000
Asthma Exacerbations (Mar et al, 2004)	790	$34,000
Minor Restricted Activity Days (Ostro & Rothschild, 1989)	1,600	$159,000

* Rounded to nearest whole number, with up to two significant figures.
** Rounded to nearest $1000.

41. Overall, these health impact counts and their dollar valuations are conservative estimates of the health benefits after the application of BART at the Navajo power plant units, as per EPA's initial proposed rule, for a number of reasons. These reasons include: (a) consideration here of health impacts only in counties within the state of Arizona, even though the air pollutant emissions from the Navajo power plant units likely also affect ambient air concentrations in other communities beyond the geographic region considered here, especially in the adjoining parts of the States of California, Nevada, Utah, Colorado, and New Mexico; (b) additional health impacts not modeled in this analysis attributable to any co-reductions in other pollutants (*e.g.*, ozone and NO_2) are not included here; (c) 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; and, (d) there are either no health impact study or no dollar valuation for many health outcomes thought to be adversely affected by air pollution, such as effects of air pollution on birth outcomes and on infants. In particular, the first draft of the most recent Nitrogen Oxides Integrated Science assessment has concluded that the respiratory effects

from short-term exposures to NO_2 are now deemed causal, while cardiovascular effects and total mortality also being likely causal (U.S. EPA, 2013). Thus, while all air pollution control costs associated with application of BART can be estimated, estimates of the health benefits and their monetary valuations are only available for a subset of likely health impacts from air pollution. This means that my analysis is conservative, and likely greatly underestimates the numbers and monetary valuations of the health benefits of applying EPA's BART determination to the Navajo power plant units.

42. Based upon my analyses, the health benefits and their valuations derived from the application of EPA's BART determination to the Navajo units are substantial. Moreover, these benefits and their valuations accrue each and every year after BART, as per EPA's rule, is operational. Accordingly, ten years from the point that BART is operational, the health benefits and valuations of BART will be roughly ten times the values provided in Table 1, before adjustment for a discount rate, as appropriate. Similarly, these benefits and their valuations are not accrued, and therefore are a cost to society, each and every year that operation of the BART, is delayed, so **even a delay of only one year carries the risk of substantial, and irreparable, harm to public health, which has substantial associated adverse economic valuations. Thus, the public health-based economic harm in Arizona from the failure to implement BART on the required timelines will be valued on the order of at least $140 million to $350 million over the subsequent 10 years, depending on the mortality study employed (and subject to adjustment for discount rate, as appropriate). Thus, it is reasonable to conclude that any additional delay to implementation of BART pollution controls at the Navajo power plant units will only exacerbate the substantial, and irreparable, harms to public health that have already been incurred to date. Delaying BART controls is at odds with the protection of public health.**

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