WRITTEN REPORT OF GEORGE D. THURSTON
REGARDING THE PUBLIC HEALTH IMPACTS OF AIR EMISSIONS FROM THE
WHEELABRATOR FACILITY

November 20, 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 <u>Science,</u> <u>Lancet,</u> <u>Thorax,</u> and <u>The Journal of the American Medical Association</u> (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. A statement of my qualifications is attached to my affidavit.

SUMMARY OF REPORT

The purpose of this report is to document the adverse human health effects that are associated with exposures to air pollutants generally, and in particular, the adverse human health effects associated with the particulate emissions from the Wheelabrator Facility in Baltimore, Md.

This report documents how emissions contribute to the serious and well-documented adverse human health effects known to be associated with exposure to air pollution. 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 air quality modeling conducted by Dr. 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 adverse human health impacts that would occur each year if the Wheelabrator plant continues its present operations and associated air emissions, as well as the annual economic valuation of those health impacts.

BACKGROUND

The adverse health consequences of breathing air pollution 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 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 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 pollution sources. 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 (um) – 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 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 shown qualitatively consistent acute cardiac effects, such as 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 PM2.5 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. (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.

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 ug/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.*"[1]

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

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

particle exposure, even after correcting for potentially confounding factors such as age, sex, race, smoking, etc.
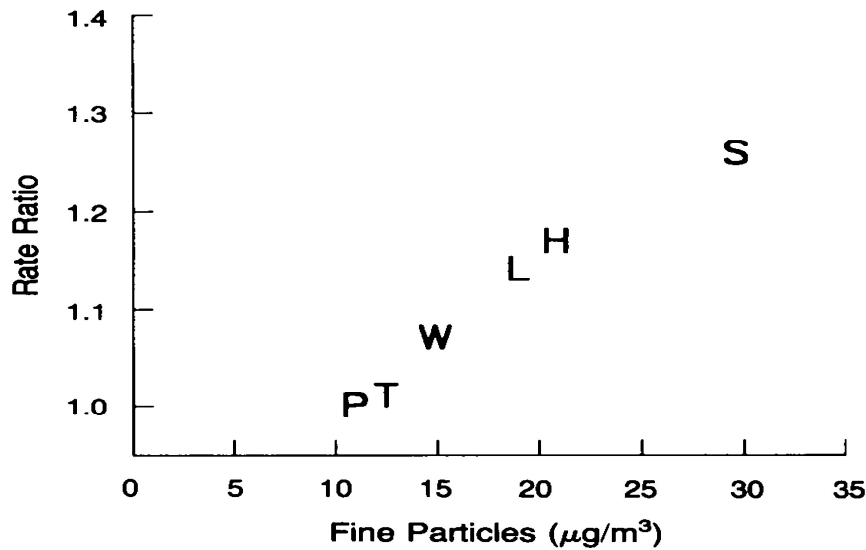

Figure 1. The Harvard Six-Cities Study showed that the lifetime risk of death increased across 6 U.S. cities as the average fine PM levels increased. (Source: Dockery et al., 1993).

In addition, a study that I wrote with co-authors, published in the Journal of the American Medical Association (JAMA), documented that long-term exposure to combustion-related fine particulate air pollution is an important environmental risk factor for cardiopulmonary and lung cancer mortality. Indeed, Figure 2 from this study indicates that the increase in risk of lung cancer from long-term exposure to $PM_{2.5}$ in a polluted city 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.
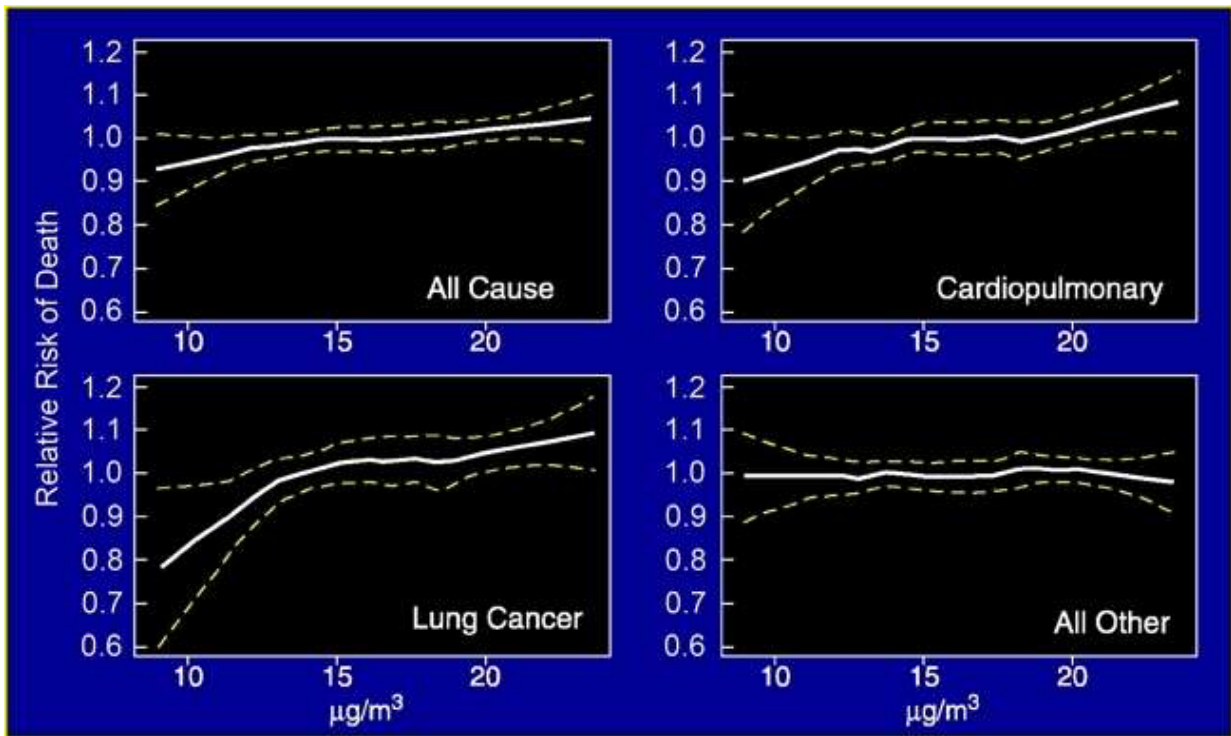
Figure 2. Cardiopulmonary and lung cancer mortality risks increase with exposure to long-term fine PM (adapted from: Pope, Burnett, Thun, Calle, Krewski, Ito, and Thurston, 2002)

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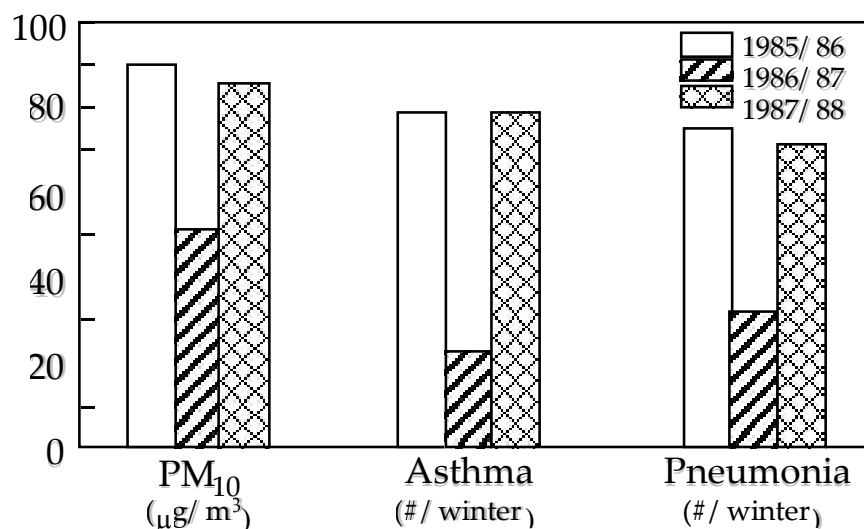
Figure 3. Decreasing PM pollution lowered the number of children's hospital admissions (Source: Pope, 1989).

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

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

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 $\mu g/m^3$.
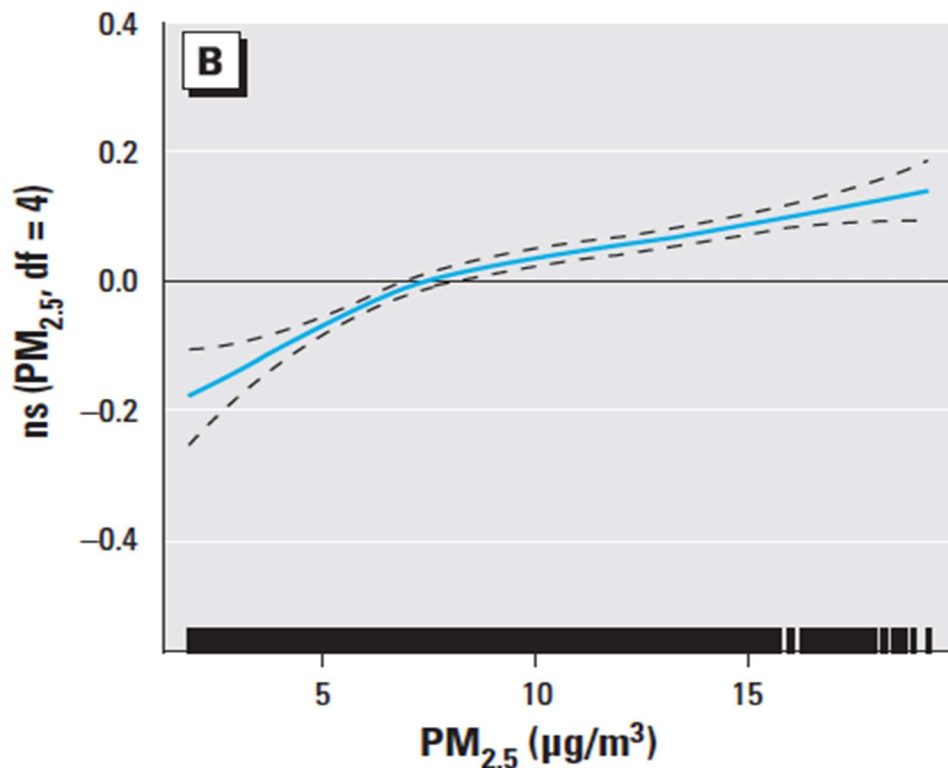

Figure 4.   Cardiovascular Mortality Risk vs. $PM_{2.5}$ exposure (solid line) and 95% CIs (dashed lines), showing increasing risk of death with increasing $PM_{2.5}$, even at very low ambient levels of $PM_{2.5}$ air pollution (from Crouse et al., 2012).

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.
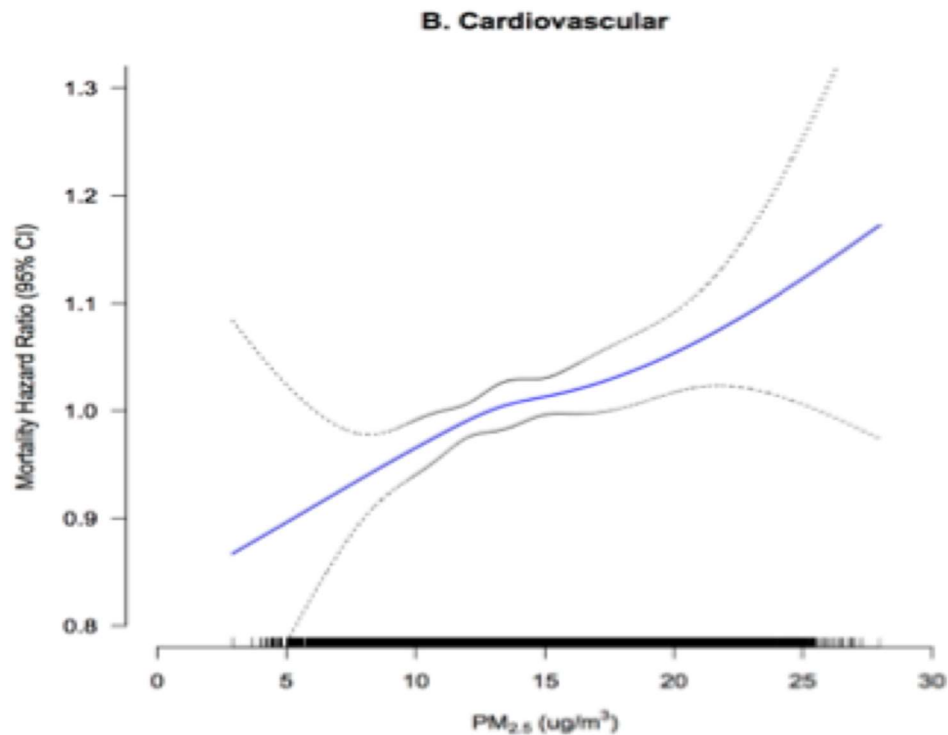
B. Cardiovascular

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.

A more recent documentation of the fact that there is a scientific consensus that air pollution effects occur even at levels below the prevailing NAAQS is the recent article from Fann et al (2017) which states very clearly: "The risk coefficient assumes a log-linear relationship between $PM_{2.5}$ and mortality over all possible values of $PM_{2.5}$, such that there is no threshold concentration below which $PM_{2.5}$-attributable mortality falls to zero. This assumption is consistent with findings in previous studies, which reported no evidence of a population-level threshold in the relationship between long-term exposure to $PM_{2.5}$ and mortality, and so we elected not to apply one in this health impact function (Crouse et al. 2012; Schwartz et al. 2008; U.S. EPA 2009)."

In addition, a very recent nationwide analysis of mortality in a cohort of all Medicare beneficiaries in the continental United States (60,925,443 persons), published in the New England Journal of Medicine, found that "In the entire Medicare population, there was significant evidence of adverse effects related to exposure to $PM_{2.5}$ and ozone at concentrations below current national standards." (Di et al, 2017).

Similarly, Perlmutt et al (2017) found in their research on the effects of air pollution on New York City hospital admissions that "The majority of excess hospital admissions (i.e., > 90% in Bronx County) occurred when the AQI was <100 ('good' or 'moderate' level of health concern) regardless of whether $PM_{2.5}$ was the driver pollutant." Since when the air quality is below 100, it is within the air quality standards, this work is consistent with my testimony, and with the general scientific consensus that air pollution levels below the National Ambient Air Quality Standards (NAAQS) do have significant adverse health impacts.

Moreover, even the World Health Organization (WHO) has also concluded that there are human health effects from air pollution exposures below the legal air quality limits. The WHO has stated that[3] "The lower the levels of air pollution, the better the cardiovascular and respiratory health of the population will be, both long- and short-term" and that "Small particulate pollution have health impacts even at very low concentrations – indeed no threshold has been identified below

[3] http://www.who.int/mediacentre/factsheets/fs313/en/ (Updated September, 2016).

which no damage to health is observed.". Thus, the WHO agrees with my assessment that that adverse human health effects from air pollution exposures occur below the legal air quality limits, and that any addition to that pollution will result in an increased risk of adverse health effects, even when below the prevailing air quality standards (WHO, 2006).

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.

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 and economic impacts of ambient air pollution (Abt Associates, 2010; U.S. EPA, 2015). Analysts have relied upon BenMAP to estimate the health impacts from air pollution 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.

BenMAP is primarily intended as a tool for estimating the health impacts, and associated economic values, associated with 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.

Key assumptions of the BenMAP model, as applied here, are that:

- the Concentration -Response (C-R) function is non-threshold, and can be extrapolated down to background concentrations;
- the C-R functions can be transferred from study location to all locations in the U.S.;

• the C-R functions only applies to population examined in study; and,

• the C-R function is constant over time and environmental conditions.

Inputs to health impact functions in this work included (as reflected in Figure 1):

• The ambient air pollution impact level (as provided by Dr. Andrew Gray, of Gray Sky Solutions). All three species concentrations were estimated by interpolating 9 km gridded CALUFF model results to the county centroids. For Baltimore City, Dr. Gray used the results of local-scale AERMOD modeling for the directly emitted fine PM (and added that to the CALPUFF-predicted secondary SO_4 and NO_3);

• pollutant health effect estimates (based upon the scientific literature, 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.

Figure 1. Overview of the BenMAP Health Effects and Valuation Methodology
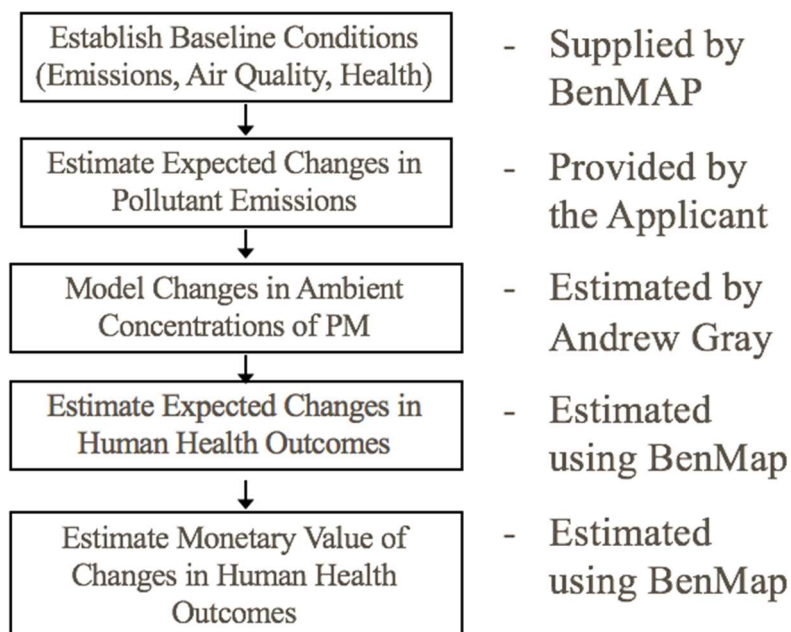
For example, in the case of a premature mortality health impact function, the BenMAP calculation can be represented, in a simplified form, as:

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

• **Air Pollution Impact**. 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, or by a specific source adding to the prevailing pollution levels (as in this case). In the case of particulate matter, this impact 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 (e.g., added to the prevailing pollution by a single source, as considered here). 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 Tables 1 and 2) BenMAP estimates for the lower end of mortality estimates (Krewski et al., 2009) (vs. higher Laden et al, 2006 and Lepeule et. al, 2012 options in BenMAP). 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 mentioned above (which are also scientifically supportable), the dollar valuation of health impact estimates in Tables 1 and 2 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.

- **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, based on Census data for each county within BenMAP.

Note that, in practice, most studies derived their estimates using a logn-linear specification. For a given health outcome y, a standard log-linear specification might look like $y=Be^{\beta PM}$., where "B" is the C- R function, and "PM" represents the pollutant concentration. This implies that the change in incidence of y is: $\Delta y = y_0(e^{\beta \, \Delta PM}-1)$.

For example, for all cause mortality:

ΔAll Cause Mortality = $y_0 \, (e^{-\beta \, \Delta PM2.5}-1) \cdot P$

Key elements: y_0 =   county-level all-cause annual death rate per person ages 30 and older

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[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 Tables 1 and 2.

$\beta =$ the pollution effect coefficient = 0.0058;

$\Delta PM_{2.5}$ = the modeled change in annual <u>mean</u> $PM_{2.5}$ concentration; and,

$P =$ total population, 30 and older

For this work, population-weighted centroid $PM_{2.5}$ concentration impacts in each county in Maryland and the surrounding six study states (Connecticut, Delaware, DC, Kentucky, Massachusetts, New Jersey, New York, Ohio, Pennsylvania, Virginia, West Virginia) were determined by Andrew Gray for the existing plant emissions.[5] Postprocessing of the CALPUFF results was performed to sum the modeled sulfate, nitrate, and $PM_{2.5}$ at each receptor, in order to obtain the estimated total fine PM concentration at each receptor. These values were entered into BenMAP to estimate the health impacts, and their dollar valuations, associated with the plant's pollution impacts on a county-by-county basis. The county-level results were then summed on a cumulative basis (Tables 1 and 2, with both numbers and valuations, by cause). Furthermore, to allow an indication of the Maryland-specific impacts of the Wheelabrator emissions, the health impact valuations (summed over all causes, as dollars) were also calculated.

RESULTS

Using the above-described EPA BenMAP methodology-based analysis, I conservatively estimate the total potential public health-based economic benefits associated with reductions in ambient $PM_{2.5}$ concentrations as a result of eliminating the air emissions from the Wheelabrator plant (as displayed in Table 1 for all seven affected states considered, combined) to be roughly $55 milion per year, overall). As also shown in Table 1, the numbers of avoidable adverse health events from the plant 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.

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[5] "Existing" emissions are actual emissions rates for the three incinerator boilers, reported in pounds per hour by Wheelabrator on Annual Emissions Certification Reports for 2014, 2015, and 2016, and converted to an average annual rate for all three boilers.

Table 1. Annual Multi-State Human Health Effects and Monetary Valuations Associated With the PM$_{2.5}$ Air Pollution Impacts from the Wheelabrator Plant

Health Endpoint	Number Per Year	Total Dollar Valuation (2010$)
Respiratory Hospital Admissions (Kloog et al., 2012; Zanobetti et al., 2009)	0.9[a]	$27,729
Cardiovascular Hospital Admissions (Bell et al., 2008; Peng et al., 2008; Peng et al., 2009; Zanobetti et al., 2009)	1.2[a]	$43,918
Acute Bronchitis (Dockery et al., 1996)	8.2	$3,919
Acute Myocardial Infarction, Nonfatal (Pope et al., 2006; Sullivan et al., 2005; Zanobetti et al., 2009; Zanobetti & Schwartz, 2006)	0.6[b]	$78,440[a]
Emergency Room Visits (Glad et al., 2012; Mar et al., 2010; Slaughter et al., 2005)	4.8[b]	$2,044
Asthma Exacerbation Symptoms (Mar et al., 2004; Ostro et al., 2001)	158.2[b]	$9,139
Upper Respiratory Symptoms (Pope et al., 1991)	148.4	$4,936
Lower Respiratory Symptoms (Schwartz and Neas, 2000)	104.0	$2,185
Minor Restricted Activity Days (Ostro & Rothschild, 1989)	4622.2	$315,414
Work Days Lost (Ostro et al., 1987)	776.3	$144,895
Chronic Bronchitis (Abbey et al., 1995)	4	$1,116,548[c]
Mortality, All Causes (Krewski et. al, 2009)	5.5	$53,051,923
Total Valuation		$54,801,090

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.

As shown in Table 2, for Maryland alone, the largest health impacts of the plant occur in the state in which the facility is operating, but, because this pollution can be carried so far downwind, nearly more than half of the plant's air pollution health impacts accrue in other (downwind) states.

Figure 2 displays the spatial distribution of the total valuation of health impacts across Maryland. Again, it is seen that the greatest potential health benefits of eliminating emissions from the plant would result in Baltimore and in the downwind counties near to Baltimore, as expected.

Table 2. Annual Maryland Human Health Effects and Monetary Valuations Associated With the PM$_{2.5}$ Air Pollution Impacts from the Wheelabrator Plant

Health Endpoint	Number Per Year	Total Dollar Valuation (2010$)
Respiratory Hospital Admissions (Kloog et al., 2012; Zanobetti et al., 2009)	0.3[a]	$10,763
Cardiovascular Hospital Admissions (Bell et al., 2008; Peng et al., 2008; Peng et al., 2009; Zanobetti et al., 2009)	0.4[a]	$16,803
Acute Bronchitis (Dockery et al., 1996)	3.0	$1,462
Acute Myocardial Infarction, Nonfatal (Pope et al., 2006; Sullivan et al., 2005; Zanobetti et al., 2009; Zanobetti & Schwartz, 2006)	0.2[b]	$29,201
Emergency Room Visits (Glad et al., 2012; Mar et al., 2010; Slaughter et al., 2005)	2.4[b]	$1,003
Asthma Exacerbation Symptoms (Mar et al., 2004; Ostro et al., 2001)	59.5[b]	$3,435
Upper Respiratory Symptoms (Pope et al., 1991)	55.3	$1,841
Lower Respiratory Symptoms (Schwartz and Neas, 2000)	38.8	$815
Minor Restricted Activity Days (Ostro & Rothschild, 1989)	1770.8	$120,838
Work Days Lost (Ostro et al., 1987)	297.6	$55,091
Chronic Bronchitis (Abbey et al., 1995)	1.5	$419,644[c]
Mortality, All Causes (Krewski et. al, 2009)	2.2	$21,160,530
Total Valuation		$21,821,425

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.

Figure 2. Map of Maryland Health Impact Valuations in Dollars, by County

Total Valuation ($, in millions)
1 2 3 4 5 6 7 8 9
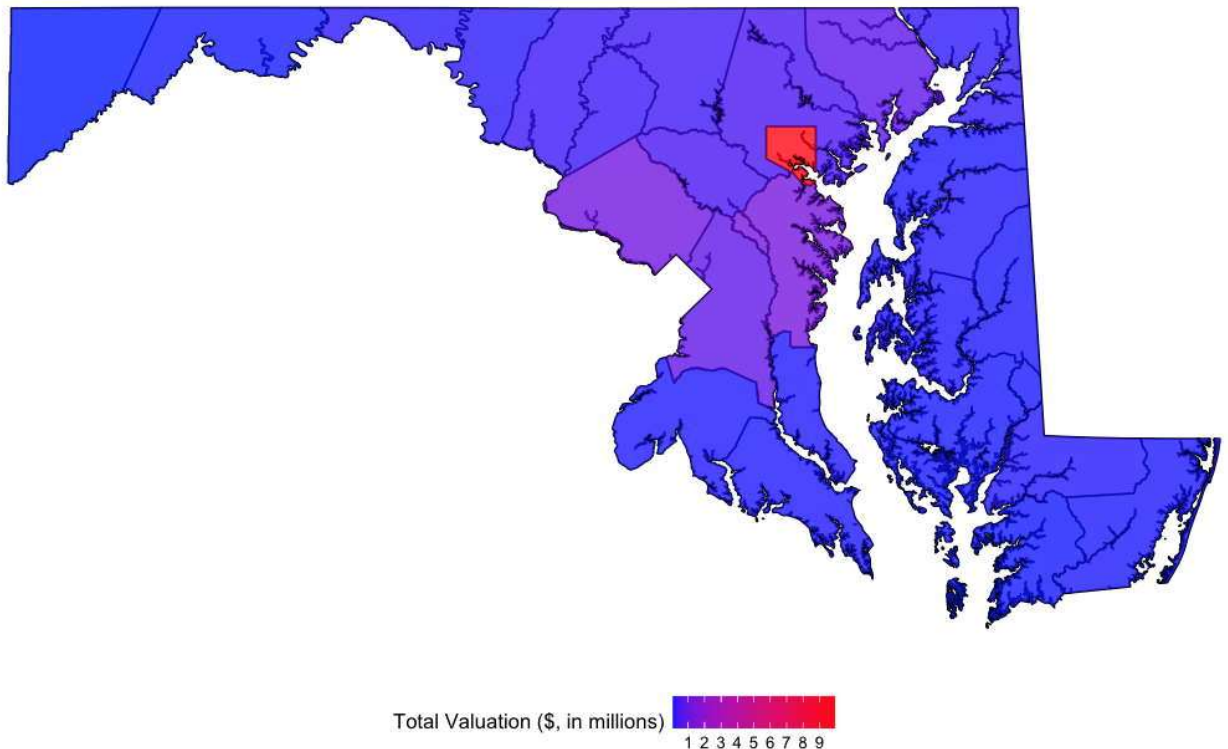
In addition to reflecting a conservative (i.e., low) mortality effects estimate, these overall health impact counts and their dollar valuations are conservative estimates of the plant's health impacts for a number of reasons, including: (a) additional health impacts not modeled in this analysis attributable 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 1 and 2 we have applied the low estimate of the mortality impacts (whereas applying the other two study options noted above would roughly double or triple the estimates in Tables 1 and 2, respectively) . Thus, these estimates of the health impacts and their monetary valuations are only available for a subset of likely health impacts from air pollution. This means that my analysis is very conservative, and likely underestimates the health and monetary impacts of the air pollution resulting from the Wheelabrator plant emissions.

CONCLUSIONS

Even applying conservative estimates and assumptions, the health impacts from the Wheelabrator plant's air emissions, and their dollar valuations, are substantial. Moreover, these impacts and their valuations accrue each and every year it is operational. Accordingly, over ten years, the health impacts and valuations will be roughly ten times the values provided in Tables 1 and 2, before adjustment for a discount rate and future affected population growth, as appropriate. Similarly, these public health impacts and their associated valuation costs continue each and every year that pollution continues. 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 in eliminating pollution from the Wheelabrator plant will only exacerbate the substantial, and irreparable, harms to public health that have already been incurred to date by the operation of this plant.

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